

Textbook of
Surgery for Dental Students

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Textbook of **Surgery for Dental Students**

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Textbook of Surgery for Dental Students

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Dedicated to Mama
The constant source of my inspiration



“Thirty years from now it won’t matter what shoes you wore, how your hair looked, or the jeans you bought. What will matter is what you learned and how you used it.”



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Preface

While teaching BDS students, I felt the need of a comprehensive and syllabus-oriented book for them. Keeping that in mind, I have written this book in a simplified manner, covering all the topics as per their revised syllabus. This textbook has primarily been directed to the students at undergraduate (BDS as well as MBBS) level. It is designed to be easy to read using a similar layout for each topic. The text is written in a short bulleted form with many headings so that the required information can be found readily. In addition, the text contains many boxes with salient points to assist students in quick revision at the end. A large number of colored clinical photographs have been incorporated in the text to make the subject more understandable. I have tried hard to make sure that the facts in the book are as accurate as possible, taking help from the specialists of various fields to either write or review the relevant sections. Another fact remains that the dental students are usually not exposed to routine ward procedures and minor surgical operations required as per their curriculum. For that, I have compiled a DVD covering stepwise description of these procedures hoping it will make the learning process a lot easier for the students.

It has been correctly said that a textbook lives through its readership. Despite the best of my efforts, some misprints or factual errors might have crept in unnoticed. I shall be grateful to the readers for giving me suggestions for improvement and pointing out mistakes that can be corrected in future.

Sanjay Marwah



Acknowledgments

I have relied on a lot of people for preparation of this text and I thankfully acknowledge their help.

Dr Sham Singla, Senior Professor of Surgery, PGIMS, Rohtak, has devoted his entire career to education of surgeons in training and is a very popular teacher amongst the undergraduate and postgraduate students. Apart from contributing chapters in this book, he has critically gone through the script multiple times and incorporated necessary corrections. I am highly thankful for his advice and guidance.

My wife Dr Nisha Marwah, Professor of Pathology, PGIMS, Rohtak, has contributed chapters in the book and has spent a lot of time in making corrections in the script. Her constant guidance, appreciation and support have helped me move forward at each step.

I would like to thank Ms Shruti Kirti, an outstanding IIIrd year BDS student at my institute, who devoted her time in proofreading each chapter and gave me valuable suggestions in simplifying the text. Also, Dr Shashi Pratap and Dr Jai Prakash, my postgraduate students, went out of the way to put in all the required last minute efforts for completion of the textbook.

I take this opportunity to thank my dear friends Dr Kulvinder S Bahl MS (Surgery), Prof Dhruv Chaudhary DM (Pulmology and Critical Care), Prof Harpreet Singh MD (Medicine) and Prof Sunita Singh MD (Pathology) for their constant assistance through all my endeavors.

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I express my gratitude to Shri Jitendar P Vij (Chairman and Managing Director) and Mr Tarun Duneja (Director-Publishing) of M/s Jaypee Brothers Medical Publishers (P) Ltd, for their encouragement and novel suggestion of adding a DVD of minor operative procedures.

I am also grateful to Mr Manoj Pahuja, Computer Art Designer, for his creative ideas that simplified the illustrations and made them more informative. I thank every member of production staff for giving this book the best possible shape and bringing it out so effectively.

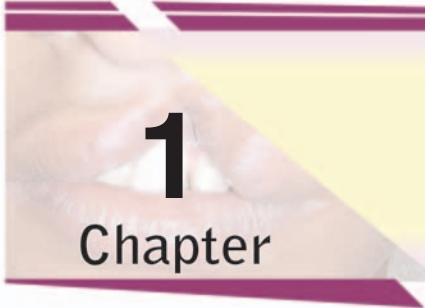
I cannot forget to mention and appreciate the efforts of Mr Atul Jain, M/s Jain Book Depot, Rohtak who encouraged me to write this book and was always available for any kind of help.



Contents

1. Introduction of Surgery	1
<i>Sanjay Marwah</i>	
2. Inflammation	8
<i>Nisha Marwah, Sanjay Marwah</i>	
3. Infections	15
<i>Sanjay Marwah</i>	
4. Specific Infections	22
<i>Sanjay Marwah</i>	
5. Sinus, Ulcer and Fistula	36
<i>Sanjay Marwah</i>	
6. Wounds	46
<i>Sanjay Marwah</i>	
7. Surgical Asepsis and Antiseptic Measures	54
<i>Sanjay Marwah</i>	
8. Hemorrhage, Blood Transfusion and Bleeding Disorders	59
<i>Nisha Marwah, Sanjay Marwah</i>	
9. Shock, Water-Electrolyte and Acid Base Balance	67
<i>Sanjay Marwah, Jasbinder Kaur</i>	
10. Care of the Acutely Injured	82
<i>Sanjay Marwah</i>	
11. Tumors	89
<i>RK Karwasra, Sanjay Marwah</i>	
12. Cysts and Neck Swellings	107
<i>Sham Singla, Sanjay Marwah</i>	
13. Diseases of Lymph Nodes and Lymphatics	127
<i>Sanjay Marwah</i>	
14. Diseases of Oral Cavity	140
<i>Sanjay Marwah</i>	
15. Diseases of Salivary Glands	161
<i>Sanjay Marwah</i>	
16. Diseases of the Larynx	175
<i>Sanjay Marwah</i>	

17. Head Injury and Cranial Nerves Injury	184
<i>Sanjay Marwah</i>	
18. Gangrene and Diseases of Arterial System	198
<i>Sanjay Marwah</i>	
19. Diseases of Venous System	220
<i>Sanjay Marwah</i>	
20. Principles of Operative Surgery, Diathermy, Radiotherapy and Anesthesia	228
<i>Sanjay Marwah, Naveen Malhotra</i>	
21. Fractures and Maxillofacial Fractures	244
<i>Sanjay Marwah, Virendra Singh</i>	
22. Cleft Lip and Cleft Palate	262
<i>Sanjay Marwah</i>	
23. The Thyroid Gland	267
<i>Sham Singla, Sanjay Marwah</i>	
24. The Parathyroid and Pituitary Gland	291
<i>Nisha Marwah, Sanjay Marwah</i>	
25. Swellings of the Jaw	299
<i>Sanjay Marwah, Virendra Singh</i>	
26. Imaging Techniques for Head and Neck Lesions	315
<i>Sanjay Marwah</i>	
27. Burns and Skin Grafting	326
<i>Sanjay Marwah</i>	
28. Surgical Suturing	336
<i>Sanjay Marwah</i>	
29. Surgical Instruments	344
<i>Sanjay Marwah</i>	
30. Wound Dressings and Bandages	363
<i>Sanjay Marwah</i>	
31. Surgical Specimens	368
<i>Nisha Marwah, Sanjay Marwah</i>	
 <i>Index</i>	 373



1

Chapter

Introduction of Surgery

Sanjay Marwah

HISTORY OF SURGERY

There have been evidences that the art and craft of surgery was developed even during prehistoric cultures.

- The human remains of Neolithic times and cave paintings show holes drilled into the skull exposing the dura mater to treat intracranial diseases.
- Early Harappan periods of Indus Valley Civilization (modern day Pakistan) show evidence of teeth being drilled during 3300 BC.
- In ancient Egypt, a mandible shows two perforations just below the root of first molar indicating drainage of tooth abscess during 2650 BC.
- Sushruta was well-known Indian physician who taught and practiced surgery on the banks of Ganges during 600 BC. He wrote volumes of surgical text books (**Susrutha Samhita**) and is known as **Father of Surgery**. His books described method of examination, diagnosis, treatment and prognosis of various illnesses. He also described detailed operative techniques of plastic and cosmetic surgery.
- In ancient Greece, Hippocrates was the Greek physician who innovated the famous **Hippocratic Oath**.
- In ancient China, Hua Huo was a famous Chinese physician who was the first to perform surgery with help of anesthesia.
- In middle ages, surgery was developed in the Islamic world. Abulcasis was a great medieval surgeon who wrote comprehensive textbooks and is often regarded as **Father of Surgery**.
- In Europe, the surgery became a formal subject and got split away from medicine in 15th century. Rogerius Salernitanus composed the modern surgical manual (**Chirurgia**) that continued up to modern times.

In 19th century, degree of bachelor of surgery (ChB) began to be awarded with bachelor of medicine (MB) that later became MBBS. The master degree became the higher degree and was awarded as master of surgery (MS).

- During world wars, the battlefield doctors became surgeons by pioneering the treatment of gunshot wounds. Naval surgeons were often barbers doing surgery as an additional job.
- The modern surgery progressed at a rapid pace based on three developments:
 - a. **Control of bleeding:** Before modern surgery developed, there was a real threat of patient bleeding to death during operation. Wound cautery with extreme heat was tried as an effort to control bleeding. But it was destructive, painful and had poor outcome. Concept of ligating the bleeding vessels was given by Abulcasis in 10th century that was much better than cautery. But it was also very dangerous because of high rate of infection caused by ligatures. Later the results of ligatures improved once the concept of infection control came in. In early 20th century, concept of blood grouping allowed effective blood transfusion.
 - b. **Control of infection:** The concept of infection control was unknown till early modern times. In 1847, Hungarian doctor Ignaz Semmelweis noticed that medical students coming from dissection hall were causing excessive maternal death compared to midwives. He introduced compulsory handwashing for everyone entering the maternal wards leading to significant decrease in maternal and fetal death. However, his advice was dismissed by Royal Society in UK.

Later, Joseph Lister, a British surgeon, started using phenol during surgery to prevent infection that quickly reduced the infection rate. He also introduced techniques of instrument sterilization, rigorous handwashing and rubber gloves for surgical procedures. He published his work in **The Lancet** in 1867 and he was named **Father of Antiseptic Surgery**.

- c. *Control of pain*: In earlier times, surgery was traumatic and very painful procedure. Control of pain or anesthesia was first discovered by two American Dental Surgeons, Horace Wells (1815-1848) and William Morton. With discovery of anesthetic chemicals (ether and chloroform), surgical practice changed dramatically. Later, discovery of muscle relaxants (curare) allowed prolonged and complex surgeries to be performed effectively.

Consequently, other developments that led to the progress of modern day surgery are:

- Development of **imaging techniques** (See Chapter 26).
- **Microvascular and reconstructive surgery**: It is aimed at reattachment of severed limbs, digits, or other body parts by plastic surgeons. Modern techniques such as the use of a bone grinder to assist in grafting bone back into place are becoming more common.
- **Transplant surgery**: In case some vital organ is damaged by disease process (kidney, liver), it is removed and replaced by the same organ retrieved from the human donor (live or cadaver). It involves complex microvascular procedures. Since transplanted organ is a foreign element to the body, it is likely to be rejected by autoimmune response. Its rejection is prevented by use of immunosuppressive drugs. Once the transplant is taken up, the patient is able to lead near normal life.
- Development of **minimal access surgery**. It is a technique that helps in performing surgical procedures with less invasion, less disfigurement, less postoperative pain and early recovery of the patient. With increasing experience, surgeons are becoming experts in performing major surgical procedures with minimal access surgery.

Various minimal access techniques are:

- i. *Laparoscopic surgery*: The peritoneal cavity is inflated with carbon dioxide to produce pneumoperitoneum. A telescope is then introduced to visualize the inside of peritoneal cavity by projecting the image on a television screen (Video assisted surgery). Various instruments are then introduced into peritoneal cavity through various ports in abdominal wall to perform the surgical procedures, e.g. laparoscopic cholecystectomy, hernia repair etc.
 - ii. *Thoracoscopic surgery*: The thoracic cavity is entered in the same way (as laparoscopy) to perform various procedures in the thoracic cavity.
 - iii. *Endoscopy*: Flexible tubes are introduced into hollow organs (esophagus, colon, urinary bladder) through natural orifices for visualization of internal pathologies and their management. ENT surgeons perform minimal access surgery on ear and paranasal sinuses using small flexible endoscopes.
 - iv. *Arthroscopy*: Visualization of inside of joint spaces, e.g. knee joint.
 - v. *Endoscopic brain surgery*: Flexible endoscope and fine instruments are introduced into cranial cavity through small holes in the skull to perform surgery on intracranial lesions.
 - vi. *NOTES (Natural Orifice Transluminal Endoscopic Surgery)*: In this new concept meant for avoiding skin incision for surgery, a flexible endoscope is introduced through natural orifices (oral cavity, anal canal, vagina etc.). Then an abdominal viscus (stomach, rectum etc.) is transgressed to enter into peritoneal cavity. With the help of video-assisted surgery, operation is performed, e.g. appendicectomy or cholecystectomy and the specimen is removed through the viscus (e.g. stomach).
- **Robotic surgery**: In place of surgeons hands, robot is used for performing a surgical procedure. The surgeon sits on a computer console and gives command to the robot for performing various surgical steps. Its advantages are:
 - a. The movements are precise and free from tremors giving high accuracy in sensitive areas.
 - b. Dexterity of movements, i.e. unlike human hands, the robot can move the instruments up to 360°.

It helps in performing surgical procedures in great depth even when space for the movements is restricted.

- **Telemedicine:** With use of internet in medical sciences, it has further improved surgical teaching and training. The surgical procedure performed at one place can be telecast live at any other place through video conferencing while operating surgeon interacts with the audience. This technique is becoming very popular and is being widely used in live operative workshops meant for training young surgeons.
- **Newer energy sources:** Apart from use of high quality electrocautery (monopolar/bipolar), newer energy sources have been devised for precise tissue cutting as well as coagulation, e.g. lasers, high frequency ultrasonic waves, harmonic scalpel, etc. (See Chapter 20). These energy sources have made the minimal access endoscopic surgery very safe and simple.

DEALING WITH A SURGICAL PATIENT

Out of all medical disciplines, surgery is a unique speciality where surgeon, who is primarily a doctor, treats the disease using surgical instruments.

The stages through which a surgical patient passes is described as **Surgical crescendo**. These are:

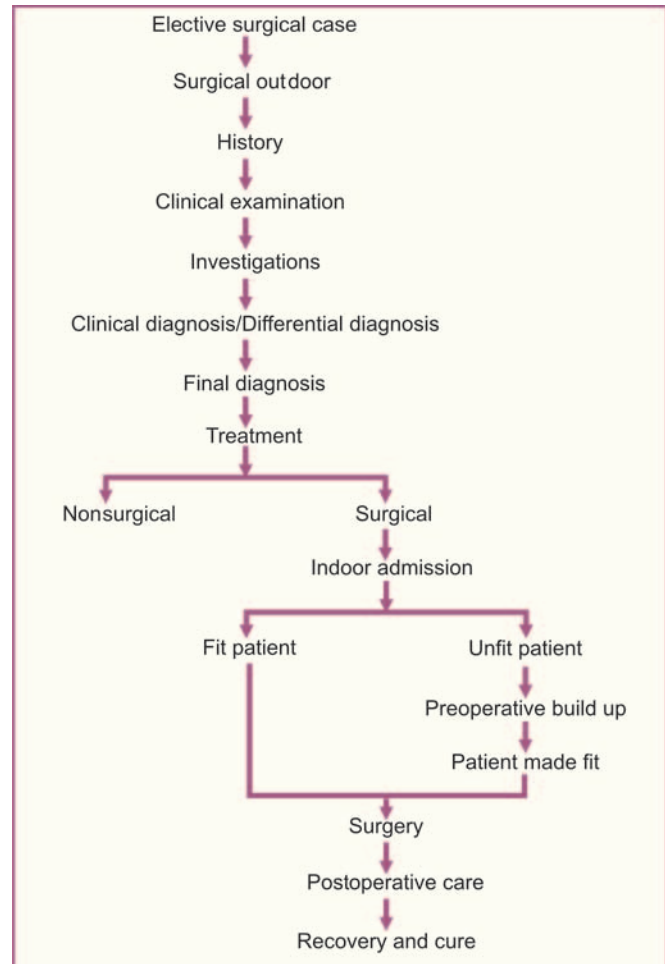
1. History taking
2. Clinical examination
3. Making clinical diagnosis and keeping possibilities of various differential diagnoses.
4. Investigations
5. Making final diagnosis
6. Surgery
7. Sometimes diagnosis is still not made even after exploration. In such situation, either nature cures the disease and diagnosis is never made or the patient dies and postmortem reveals the exact pathology.

A surgical patient coming to the hospital can be:

- Elective case
- Emergency case

Elective case reports in the surgical outdoor during routine hours where diagnosis of disease is made. Then he is admitted in indoor and operation is performed (Box 1.1).

Box 1.1: Outlines of management of elective surgical case

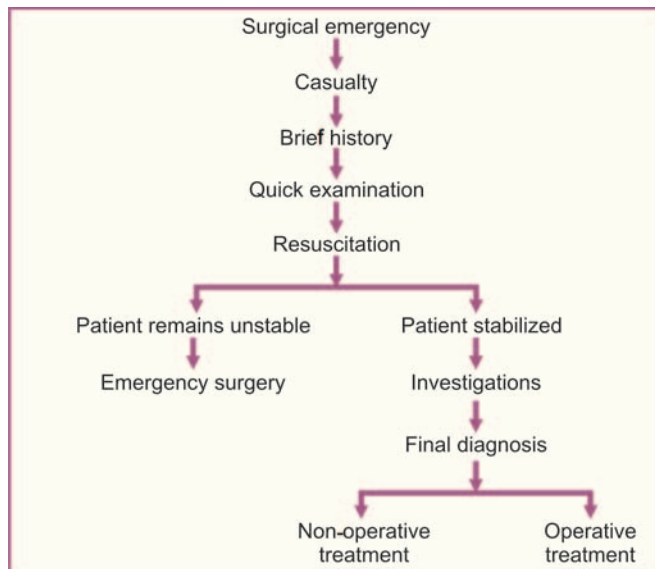


Emergency case reports in the casualty at any time and is managed in a different way. After quick history and examination, resuscitation is started.

Performing investigations and reaching the final diagnosis is considered only after the patient is stabilized. If patient remains unstable despite resuscitation, then emergency surgery is planned (Box 1.2).

It is very important to do repeated clinical examination in emergency because it helps in delineating the ongoing changes and reaching the diagnosis.

Thus, it is apparent that actual operation is only a part of total surgical care. Equally important are making diagnosis, preoperative build up and post-operative management. If diagnosis is incorrect, it may lead to improper surgery and patient may not have any

Box 1.2: Outlines of management of emergency surgical case

benefit from it. Inadequate preoperative build up can lead to intraoperative and postoperative complications and mortality. Similarly, lack of postoperative care can lead to serious postoperative complications like hypostatic pneumonia, deep vein thrombosis, wound sepsis etc.

HISTORY TAKING

History taken in outdoor or emergency relates to the specific complaints of patient so as to reach the diagnosis.

On the other hand, history taken in the indoor for admitted patient has two objectives:

- To reach the clinical diagnosis
- To look for fitness of patient for surgery.

'Symptoms' are the complaints told by the patient while **'signs'** are the features seen by the clinician on clinical examination.

Before asking 'symptoms', background of the patient is recorded that includes:

Name
Age
Sex
Marital status
Occupation
Address

Then symptoms are asked and recorded preferably in patient's own words and in 'chronological order' of their appearance.

History of Present Illness

Duration of illness It is very important to ask 'When were you perfectly well before the present illness'? The patient is likely to tell about mild episodes of similar illness in the past which otherwise he may ignore to mention. However, in reality, it may be of great importance in making the diagnosis.

Mode of onset: How the illness started, e.g. a swelling appearing on scalp after trauma is likely to be a hematoma.

Progress: Whether illness is improving or worsening, e.g. an inflammatory pathology is likely to improve with analgesics and anti-inflammatory drugs.

Aggravating and relieving factors, e.g. an inflammatory pathology is likely to be aggravated with movement of the part and relieved with rest and analgesics.

Constitutional symptoms are those which occur secondary to the illness, e.g. pain, fever, cough, nausea, vomiting, weight loss, anorexia.

Past history: Any illness suffered in the past is recorded in chronological order. It may or may not be related to present illness.

Personal history: Smoking, dietary habits, alcoholism are enquired. Marital status of the patient is asked and if married, number of children and their health is recorded. If some child has died, age and cause of death is noted.

Menstrual history: It is asked in female patients. Age at menarche, any menstrual irregularity, vaginal discharge, age at menopause, postmenopausal bleeding, etc. are recorded.

Family history: Whether any family member has suffered from similar illness. It can help in finding out genetic disorders (hemophilia) and communicable diseases (tuberculosis).

Treatment history: Any treatment taken and its effect on illness may help in reaching the diagnosis, e.g. a neck swelling improved with tablet eltroxin will suggest goiter. Any history of drug allergy and previous operations is also recorded.

EXAMINATION

General Physical Examination

Make the patient sit or lie in the bed comfortably. Examine the patient with warm hands.

Look for:

- General appearance, viz.
 - ☐ Level of consciousness (decreased in head injury).
 - ☐ Patient cooperative/uncooperative.
 - ☐ Patient anxious/lying comfortably in bed.
- Build (assessed by skeletal frame work). Skeletal deformities may be seen on exposure (Fig. 1.1).
- Nourishment (assessed by triceps skin fold thickness, subcutaneous fat, skin texture, muscle mass).
- Pulse rate (normal 72/min.), regularity, volume.
- Blood pressure (normal 120/80 mm Hg).
- Temperature (normal 37°C).
- Respiratory rate (normal 12-16/min.), regularity, type (abdominal or thoracic).
- Look for various clinical signs from head to toe:

Anemia in palpebral conjunctiva, nailbeds, tongue (areas rich in capillaries).

Jaundice in upper sclera, undersurface of tongue, palmar creases (these areas are rich in connective tissue and bilirubin has great affinity for such areas) (Figs 1.2 and 1.3).

Cyanosis Bluish discoloration of tongue (central cyanosis), bluish discoloration of tip of nose, fingers (peripheral cyanosis).

Clubbing Drumstick appearance of fingers and toes (Fig. 1.4).



Fig. 1.2: Jaundice seen in upper sclera



Fig. 1.3: Jaundice seen on undersurface of tongue

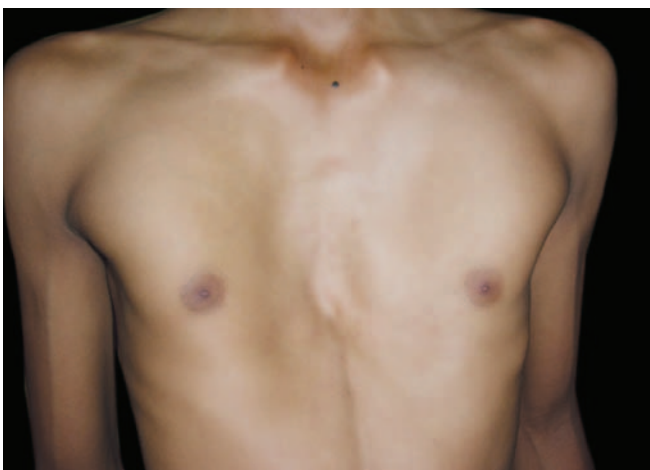


Fig. 1.1: Pigeon chest deformity



Fig. 1.4: Finger clubbing

Tracheal deviation: Normally, trachea is centrally placed in suprasternal notch.

Edema feet: Apply pressure with thumb for 10-15 seconds on the shin. Sign is positive if pit is produced at the site of pressure and remains for more than 30 seconds. Edema gives rise to soft pitting while if pus is present, induration is always felt.

Local Examination

- Side of the lesion (right or left) should always be recorded carefully.
- A few simple instruments are necessary as 'armamentarium' during patient examination. There are:
Pocket torch
Black paper
Tongue depressor
Metal scale
Measuring tape
Hammer
Stethoscope
Disposable gloves
Thermometer
Skin marking pen
- While examining a lesion, be particular in noting its site, external appearance, possible anatomical organ of origin and its effects on surrounding structures.
- The lesion may present as swelling, ulcer, sinus or fistula. Their details of examination are described in the relevant sections.
- Always examine the lymph nodes draining the site of lesion.

Systemic Examination

The aim is to know the patient as a whole. During this, some other pathology related or unrelated to presenting disease may be discovered. Various systems examined are:

CVS
CNS
Respiratory system (chest)
GIT (Abdomen)
Genitourinary system

Clinical Diagnosis

On the basis of history and examination, a clinical diagnosis is made. Aim is to localize the organ of origin, type of pathological process and its extent.

Pathological diseases are broadly classified as:

Congenital
Inflammatory (Acute or chronic)
Neoplastic (Benign or malignant)
Traumatic

Other rare ones are:

Degenerative diseases
Metabolic diseases
Hormonal diseases

In case, the diagnosis is doubtful, other possibilities are kept as differential diagnosis, starting with most probable diagnosis as first. Based on 'law of probability', commonly seen disease should be kept as first possibility.

INVESTIGATIONS

Aims of doing investigations are:

- To reach final diagnosis
- To look for fitness for anesthesia and surgery.
- In case of malignancy, staging of disease so as to plan treatment and assess prognosis.

Various investigations are decided according to the site and nature of pathology. These are:

Hematological Investigations

- Hemoglobin—for anemia.
- Bleeding time, clotting time—for bleeding disorders.
- Total and differential leucocyte count—raised in infections.
- ESR—raised in chronic infections.
- Blood Sugar—raised in diabetes.
- Blood urea and serum creatinine—raised in renal failure.
- Thyroid function tests—in case of thyroid pathology.
- Liver function tests—deranged in liver dysfunction.

Urine examination: For albumin, sugar and microscopy.

Stool examination: For ova, cyst, pus cells, occult blood.

Imaging

- X-ray—for bony changes, fractures.
- Ultrasound—differentiates solid and cystic lesions.
- Doppler imaging—for blood flow.
- CT Scan with contrast enhancement—for solid organs.
- MRI—for joints, spine.

Pathological Examination

- Fine needle aspiration cytology
- Tissue biopsy
- USG/CT guided biopsy—helpful in localizing the site of lesion especially if it is deep seated.

MANAGEMENT OF UNFIT PATIENT

- The patient should be hospitalized and built up for surgery.
- In case of severe anemia, fresh blood transfusions are given to improve hemoglobin. The patient should have hemoglobin level of 10 gm% at the time of surgery.

- In case of severe hypoproteinemia (Serum albumin < 2 gm %), parenteral nutrition should be given.
- In uncontrolled diabetes, insulin injections are given.
- In patients with chronic lung disease, preoperative preparation helps in preventing postoperative respiratory complications. Measures taken are:
Smoking cessation
Chest physiotherapy
Bronchodilators
Antibiotics (for purulent sputum)
- Uncontrolled hypertension is treated with anti-hypertensive drugs.

RISK ASSESSMENT OF THE SURGERY

Before subjecting the patient to surgery, always put following questions to yourself:

- What is the risk of surgery?
- Whether patient will benefit from the operation?

Based on risk-benefit ratio, the patient should be counseled and written consent should be obtained from him before performing the operation.

2

Chapter

Inflammation

Nisha Marwah, Sanjay Marwah

- It is defined as local response of living tissues to injury due to any agent.
- It is the response of body defense mechanisms to limit or eliminate the agent causing injury.
- It mainly affects vascular and connective tissues.
- Agents causing inflammation are:
 1. *Physical agents* Heat, cold, radiation, trauma.
 2. *Chemical agents* Organic and inorganic poisons.
 3. *Infective agents* Bacteria, virus, parasites, fungi.
 4. *Immunological agents* Cell mediated and antigen-antibody reactions.

TYPES OF INFLAMMATION

Acute Inflammation

It is of short duration. There is early body reaction followed by repair.

Chronic Inflammation

It is of longer duration. Either agent causing acute inflammation persists for a long time or stimulus is such that it causes chronic inflammation from the beginning.

Signs of Inflammation

Classical signs of inflammation are:

- Redness (Rubor)
- Heat (Calor)
- Swelling (Tumor)
- Pain (Dolor)

These four signs were described by Celsus in 1st century AD.

- *Loss of function (Functio laesa)* This fifth sign was later added by Virchow.

These changes are typically more prominent in acute inflammation than in chronic inflammation.

Acute Inflammation

The acute inflammation has two main components:

1. Vascular events
2. Cellular events

Vascular Events

It includes:

- a. Changes in vascular flow and caliber
 - b. Increased vascular permeability
- a. *Changes in vascular flow and caliber:* These are characterized by following sequence of events:
 - Transient vasoconstriction for a few seconds.
 - Next follows persistent progressive vasodilatation involving mainly arterioles. This results in increased blood flow to the area and is responsible for heat and redness.
 - Progressive vasodilatation may elevate local hydrostatic pressure resulting in transudation.
 - Next occurs slowing or stasis of microcirculation.

These hemodynamic changes are best explained by Lewis's triple response that includes a "flush", a "flare" and a "wheal".

- The **flush** appears immediately following stroking as a dull red line and is due to capillary dilatation.
- The **flare** is a bright red irregular surrounding due to arteriolar dilatation.
- The **wheal** is a swelling or edema of surrounding skin occurring due to transudation of fluid into extravascular space.

- b. *Increased vascular permeability*: During inflammation, endothelium lining of microvasculature becomes more leaky resulting in escape of protein rich fluid into the interstitial compartment and this fluid is known as exudate.

Thus, edema in initial stages is due to increased hydrostatic pressure (transudation) while in later stages it is due to increased vascular permeability (exudation).

Cellular Events

It consists of two processes:

- a. Leukocyte extravasation
 - b. Phagocytosis
- a. *Leukocyte extravasation*: The escape of leukocytes from the lumen of microvasculature to the interstitial tissue is the most important feature of the inflammatory response. In acute inflammation, neutrophils reach the site of injury first followed by monocytes and macrophages.
- Steps of leukocyte extravasation include:
- Stasis of blood and changes in axial flow of blood.
 - Margination of leukocytes and pavementing.
 - Rolling and adhesion to endothelium.
 - Emigration through inter-endothelial gaps by ameboid movements into extravascular space. Red cells also escape by passive movements (Diapedesis).
 - Chemotaxis: It is movement of leukocytes towards the site of injury and is defined as locomotion oriented along a chemical gradient.
- b. *Phagocytosis*: It is the process by which polymorphs and macrophages ingest microorganisms and other foreign particles. It is similar to feeding process of amoeba and involves following steps:
- Recognition and attachment.
 - Engulfment.
 - Killing and degradation.

CHEMICAL MEDIATORS OF INFLAMMATION

These are large number of endogenous compounds which enhance vascular permeability and also mediate other processes of acute inflammation as well including vasodilation, adhesion, chemotaxis, phagocytosis, tissue destruction and systemic effects such as fever and pain. These mediators are divided into two groups:

1. Mediators derived from cells
2. Mediators derived from plasma

Important groups of mediators, their source and action are given in Box 2.1.

SYSTEMIC INFLAMMATORY RESPONSE

If injury is severe, then apart from local inflammatory response, there is **systemic response** as well leading to *neuroendocrine, immunological and metabolic alterations*.

Endocrine Response

There is increased release of hormones namely: ACTH, cortisol, growth hormone, epinephrine, norepinephrine, glucagon, renin and aldosterone.

Metabolic Response

- a. There is increased lipolysis resulting in elevated levels of plasma fatty acids and glycerol.
- b. There is increased nitrogen excretion leading to rise in blood urea levels.
- c. There is increased production and decreased utilization of glucose by tissues leading to hyperglycemia.

Immune Response

The immune response to injury has two broad components:

Innate Response

It occurs early and is not antigen specific. It depends on functioning of natural killer (NK) cells.

Acquired Response

It occurs later after antigen processing and clonal expansion of T- and B-cells. It is antigen specific.

During innate response to injury, certain mediators are released by immunocytes. These mediators are small proteins or lipids and are known as **Cytokines**. Unlike hormones, they are not stored as preformed molecules.

The cytokines appear very rapidly after injury, bind to specific cell receptors and exert their influence by pro-inflammatory or anti-inflammatory response. Cytokine response following injury includes fever, tachycardia, leukocytosis and hyperventilation and is referred as **systemic inflammatory response syndrome**

Box 2.1: Chemical mediators of inflammation

Mediator	Source	Action
CELL DERIVED		
Vasoactive amines		
Histamin	Mast cells	Increased vascular permeability
Serotonin	Platelets	
Arachidonic acid metabolites		
Prostaglandins	Inflammatory cells Mast cells Membrane phospholipids	Vasodilation, pain, fever
Leukotrines	—do—	Leukocyte adhesion, increased vascular permeability, bronchoconstriction, vasoconstriction
Lysosomal enzymes	Neutrophils, Monocytes	Bacterial degradation, tissue damage
Platelet activating factor	leukocytes, mast cells	Platelet activation, increased vascular permeability, vasoconstriction, bronchoconstriction
Nitric oxide	Macrophages, endothelium	Vasodilation, Cytotoxicity
Oxygen metabolites	Leukocytes	Tissue damage
Cytokines and chemotrines	Leukocytes, endothelium	Endothelial effects, fibroblastic proliferation, systemic effects
PLASMA DERIVED		
Fibrin degradation products	Clotting and fibrinolytic products	Increased vascular permeability
Bradykinin	Kinin system	Increased vascular permeability, pain
Anaphylatoxins (C_{3a}, C_{5a})	Complement system	Increased vascular permeability leukocyte adhesion
C _{3b}	—do—	Chemotaxis, opsonization

(SIRS). Excess production of proinflammatory cytokines is responsible for hemodynamic instability (shock) and metabolic derangements (muscle wasting).

Pre-existing cytokine production can cause **multiple organ failure (MOF)** and mortality.

Excess production of anti-inflammatory cytokines may make the patient immuno-compromised leading to high chances of infection.

The functions of various cytokines overlap with each other.

Various cytokines related to inflammatory response are:

Tumor Necrosis Factor-alpha (TNF- α)

- It is most potent mediator of inflammation.
- It is synthesized by macrophages.
- It is earliest to appear and lasts for short time.
- It is main mediator of endotoxic shock.

- It induces muscle catabolism and causes cachexia during stress.
- It is primarily involved in programmed cell death (apoptosis).

Interleukin-1 (IL-1)

- It is also extremely potent mediator of inflammation.
- It is synthesized by wide variety of cell types including macrophages.
- It has synergistic effect with TNF- α .
- It induces classic inflammatory febrile response.
- It also induces anorexia and cachexia.
- IL-1 and TNF- α together release other inflammatory mediators. It has been seen that blocking the production of TNF- α and IL-1 helps in controlling excessive inflammatory response.

IL-6, IL-8, IL-11

These are other proinflammatory cytokines.

IL-4, IL-10 and IL-13

- These are anti-inflammatory cytokines produced by T-helper cells.
- They modulate the production and effects of TNF- α and IL-1.
- Their excess production can lead to immunosuppression, increased risk of infection and death due to sepsis.

Interferon- γ (IFN- γ)

- It has central role in innate immune response to microbial invasion.
- It activates circulating and tissue macrophages.
- It may induce acute lung inflammation due to activation of alveolar macrophages.

Inducible Nitric Oxide Synthase (NOS-2) and Cyclooxygenase-2 (COX-2)

- Action of proinflammatory cytokines is due to expression of enzymes NOS-2 and COX-2.
- NOS-2 causes vasodilatation, increased vascular permeability and inhibits platelet aggregation.
- COX-1 and COX-2 help in production of prostaglandins.
- COX-1 helps in preservation of gastrointestinal mucosal integrity.
- Pharmacological inhibition of cyclo-oxygenase activity is the basis of anti-inflammatory action of non-steroidal anti-inflammatory drugs (NSAIDs).
- However, these drugs cause gastric ulceration due to inhibition of COX-1.
- Thus, COX-2 has been identified as “inflammatory” isoform of COX and drugs causing its selective inhibition do not cause gastric ulceration.

THE INFLAMMATORY CELLS**Neutrophils**

- 40-75% of circulating leukocytes.
- Their number increases during acute bacterial infections (Neutrophilia).
- Their functions are:
 - ☐ Phagocytosis of microorganisms.
 - ☐ Engulfment of non-microbial material.
 - ☐ Destruction of basement membrane of small blood vessels and glomeruli (harmful effect).

Eosinophils

- 6% of circulating leukocytes.
- Increased number of eosinophils (eosinophilia) is seen in:
 - ☐ Allergic conditions
 - ☐ Skin diseases
 - ☐ Parasitic infections

Basophils

- 1% of circulating leukocytes.
- Role in immediate and delayed type of hypersensitivity.

Lymphocytes

- 20-40% of circulating leukocytes.
- Also present in spleen and lymphoid tissues.
- B-lymphocytes help in antibody formation
- T-lymphocytes play role in cell mediated immunity.
- Their level increases in blood in chronic infection like tuberculosis (Lymphocytosis).
- In tissues, lymphocytes are dominant cells in chronic inflammation.

Plasma Cells

- Normally not seen in peripheral blood.
- They develop from lymphocytes and are rich in γ -globulin.
- Their number is increased in:
 - ☐ Multiple myeloma
 - ☐ Hypersensitivity states
 - ☐ Chronic infections like tuberculosis

Macrophages

- These are derived from reticulo-endothelial system.
- Their functions in inflammation are:
 - ☐ Phagocytosis of foreign particles.
 - ☐ Release of enzymes (Proteases) that degrade collagen material.
 - ☐ Release of cytokines (Interleukin-I, tumor necrosis factor).
 - ☐ Release of chemotactic agents.
 - ☐ Activation of fibrinolytic system.
 - ☐ Release of coagulation factors.

Giant Cells

When macrophages fail to remove foreign particles, they fuse together to form multinucleated giant cells, e.g.

Foreign Body Giant Cells

These are cells containing multiple uniform nuclei scattered throughout the cytoplasm. These are seen in tuberculosis, chronic infections.

Langhans' Giant Cells

The nuclei are arranged at periphery to form a 'horse-shoe' appearance. These are seen in tuberculosis, sarcoidosis.

Reed-Sternberg Cells

Mirror image nuclei in the cell. These are seen in Hodgkin's lymphoma.

SPECIAL TYPES OF ACUTE INFLAMMATION

Inflammation is indicated by adding the suffix "**itis**" to the Latin name of the organ or tissue involved, e.g.

Glossitis	Inflammation of tongue
Gingivitis	Inflammation of gum
Osteomyelitis	Inflammation of bone

A few morphological types of acute inflammation are:

Catarrhal Inflammation

It is a surface inflammation associated with greatly increased secretion of clear mucus, e.g. common cold.

Pseudomembranous Inflammation

It is inflammatory response of mucosal surface (oral cavity, respiratory mucosa) to toxins or irritant gases. A membranous film forms on mucosal surface that consists of necrosed epithelium and fibrin, e.g. diphtheria.

Exudative Inflammation

Various types are based on the nature of inflammatory exudates:

- Serous inflammation:** There is excessive clear watery fluid with variable protein contents and no fibrin, e.g. blister formation in burns.
- Fibrinous inflammation:** The fibrin content of exudative fluid is high. The presence of solid fibrin leads to adhesion formation and tends to inhibit resolution, e.g. fibrinous effusion following pneumonitis.

c. **Suppurative inflammation (abscess):** It is caused by infection with pyogenic bacteria. There is tissue necrosis and formation of purulent exudate. A cavity is formed that contains pus, e.g. boil, carbuncle.

d. **Hemorrhagic inflammation:** Due to severe inflammation, there is actual rupture of blood vessels leading to hemorrhage in the exudates, e.g. hemorrhagic pneumonia due to influenza.

However, overlap of various types of exudates is common, e.g. serohemorrhagic, mucopurulent.

Ulceration

When surface epithelium of an organ or tissue is lost due to necrosis and replaced by inflammatory tissue. Common sites are skin, GIT. The ulcer can be *inflammatory* or *malignant*.

The inflammatory ulcer usually heals with treatment. However, if irritation (bacterial infection, trauma) continues, it leads to chronic and non-healing ulcer.

Details of ulcer are given in chapter 5—Sinus, fistula and ulcer.

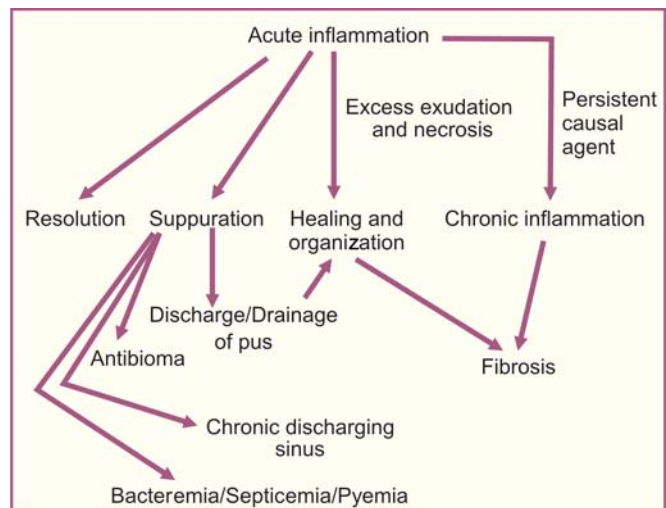
Outcome of Acute Inflammation

Acute inflammation can have following outcome (Box 2.2):

Resolution

It means complete restoration of normal tissues after acute inflammation.

Box 2.2: Outcome of acute inflammation



The factors favoring resolution are:

- Minimal cell death.
- Rapid elimination of offending organism.
- Local conditions favoring removal of fluid and debris.

Example: Resolution of lobar pneumonia.

Suppuration

There is formation of pus due to infection by pyogenic bacteria, e.g. staphylococcal infection. The superficial abscess usually ruptures spontaneously through skin or drained surgically. The swelling subsides, cavity collapses and fibrosis occurs leading to healing with scar formation. However, deep seated abscess, if not drained and treated with antibiotics only, may get organized by dense fibrous tissue forming **Antibioma** (see chapter 3—Infections) or may get calcified. Sometimes it discharges pus via a long tortuous track that fails to discharge all the abscess contents (**Chronic discharging sinus**). Sometimes, organism causing suppuration is fulminant and body defenses are weak, then it may lead to systemic sepsis in form of **bacteremia, septicemia and pyemia** (see chapter 3 Infections).

Healing and Organization

It takes place when tissue destruction in acute inflammation is excessive and there is no tissue regeneration. It leads to healing by fibrosis.

Chronic Inflammation

If causal agent is not removed, the acute inflammation may progress to chronic inflammation. In this process, inflammation and healing proceed side by side.

Treatment of Acute Inflammation

- Analgesics and anti-inflammatory drugs to control pain and swelling, e.g.
 - Non-steroidal anti-inflammatory drugs like diclofenac sodium.
 - Chymotrypsin, trypsin.
- Rest to the affected part.
- Elevation of affected part to relieve edema.
- Local application of Magnesium sulphate based ointment is hygroscopic and reduces edema of skin and subcutaneous tissues.

- Treatment of underlying cause, e.g. antibiotics for bacterial infection.
- Once abscess forms, it needs surgical drainage (see chapter 3—Infections).

CHRONIC INFLAMMATION

It is defined as prolonged process in which tissue destruction and healing continues side by side.

It can occur in following ways:

Secondary to Acute Inflammation

When causal agent of acute inflammation is not removed, it can lead to chronic inflammation, e.g. chronic osteomyelitis.

Primary to Chronic Inflammation

The causal agent is of low pathogenicity and leads to chronic inflammation from the beginning, e.g. *Mycobacterium tuberculosis* infection.

Pathological Features of Chronic Inflammation

- Infiltration by mononuclear cells.
- Presence of tissue macrophages, epithelioid cells (modified macrophages) and multinucleated giant cells.
- Tissue necrosis, e.g. central caseation necrosis in tuberculosis.
- Proliferation of granulation tissue comprising blood vessels and fibroblasts.
- Collagen formation and healing by fibrosis.

Types of Chronic Inflammation

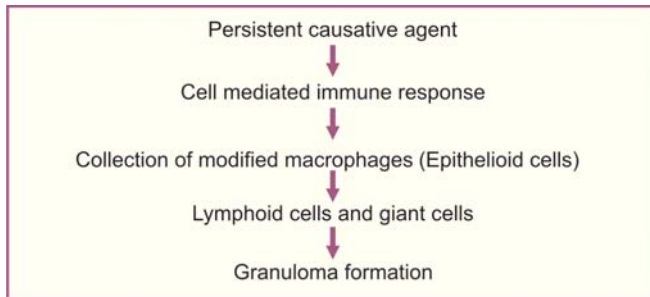
Chronic Nonspecific Inflammation

When irritant substance produces nonspecific inflammation with formation of granulation tissue and healing by fibrosis, e.g. chronic osteomyelitis.

Chronic Specific Inflammation

When the causative agent leads to characteristic histological tissue response like 'granuloma formation' (Box 2.3). It is also called as chronic granulomatous inflammation. The granuloma is a circumscribed tiny lesion about 1 mm in diameter. It consists of epithelioid

Box 2.3: Mechanism of granuloma formation



cells, lymphoid cells and giant cells along with necrosis and fibrosis. Examples are: tuberculosis, leprosy, syphilis, actinomycosis. (Details in Chapter 4, Specific infections).

3

Chapter

Infections

Sanjay Marwah

DEFINITION

Tissue invasion by organisms following breakdown of local and systemic host defenses is defined as infection. Various **host defenses** against infection are shown in Box 3.1. These defenses are lowered by multiple factors (Box 3.2).

Box 3.1: Host defenses

- Skin
- Mucosa
- Humoral immunity (Antibodies)
- Cellular immunity (Macrophages, Polymorphs, Lymphocytes)

Box 3.2: Risk factors for increased infection

Systemic	Malnutrition Metabolic (uremia, diabetes, jaundice) Lowered immunity (steroids, chemotherapy, cancer, AIDS) Shock
Local	Poor vascularity Neuropathy Poor surgical technique (Necrotic tissue, hematoma, dead space)

PATHOPHYSIOLOGY

Once bacteria invade the tissues, they release various toxins. These toxins act on macrophages which in turn release various cytokines, e.g. Interleukin-6, tumor necrosis factor (TNF), etc. These cytokines are responsible for causation of systemic manifestation of sepsis (See Chapter 2—Inflammation). Various signs and symptoms of sepsis are:

- Hyper/Hypothermia
- Tachycardia

- Hypotension
- Leukocytosis

Appearance of these manifestation in a case of sepsis is called as **Systemic Inflammatory Response Syndrome (SIRS)**. The infection can spread to local areas or systemic circulation (Box 3.3). If it remains uncontrolled, it leads to one or more organ dysfunction involving respiratory system, cardiovascular system, renal system and central nervous system. This stage is called as **Multiple Organ Dysfunction Syndrome (MODS)**. If still not controlled, it leads to **Multiple System Organ Failure (MSOF)** and death (Box 3.4).

Box 3.3: Spread of infection

- Local spread — Cellulitis
- Regional spread — Lymphangitis, Lymphadenitis
- Systemic spread — Blood (Bacteremia, Septicemia)
Body cavities (Meningitis, Peritonitis)

Box 3.4: Progress of uncontrolled infection

Infection → SIRS → MODS → MSOF → Death

WOUND INFECTION

- It is defined as collection of pus in the wound that is discharged spontaneously or requires surgical drainage.
- Wound infection is called **major infection** if it has associated systemic manifestations of SIRS.
- If systemic manifestations are not there, it is called as **minor wound infection**.
- Source of wound infection can be **endogenous** or **exogenous**.

- The exogenous infection is usually hospital acquired infection and is known as **Nosocomial infection** (Box 3.5).

Box 3.5: Source of nosocomial infection

- Surgeon's hands
- Patient's skin
- Surgical instruments
- Contaminated air

- To prevent wound infection, apart from aseptic measures, prophylactic antibiotics are used to kill the bacteria. However, following trauma/surgery, host defenses do not start in initial 4 hours. Hence, ideal time for giving prophylactic antibiotics is at induction of anesthesia so that antibiotic levels in blood and tissues are maximum during surgery.
- Once infection is established, the treatment is drainage, regular dressing and antibiotics according to culture and sensitivity report of the pus.

Common types of infection are:

BOIL (FURUNCLE)

It is the abscess in sweat gland or hair follicle (Fig. 3.1). It is caused by *Staph aureus*. There is intense inflammatory reaction leading to tissue necrosis and formation of central core of pus. It is surrounded by a peripheral zone of cellulitis. The patient complains of acute onset swelling with throbbing pain. There are



Fig. 3.1: Boil



Fig. 3.2: Abscess cheek

Box 3.6: Complications of boils

- Necrosis and sloughing of skin
- Scarring
- Excruciating pain in external auditory canal
- Cavernous sinus thrombosis in boil upper lip and nose ('dangerous area')
- Abscess leading to pyemia and septicemia

usually no systemic features of sepsis. Most of the times, overlying skin undergoes necrosis and small pustule gets drained spontaneously. If the boil subsides without suppuration, it is called 'blind boil'.

Boil of external auditory canal is extremely painful because skin is adherent to underlying cartilage and there is no space for expansion (Box 3.6).

In case of intense pain and inflammation, antibiotics (Cloxacillin), anti-inflammatory and analgesics are given along with local antiseptic application.

Sometimes incision and drainage is required if boil is big sized and not resolving with antibiotics.

In case of recurrent boils, diabetes should be ruled out.

ABSCCESS

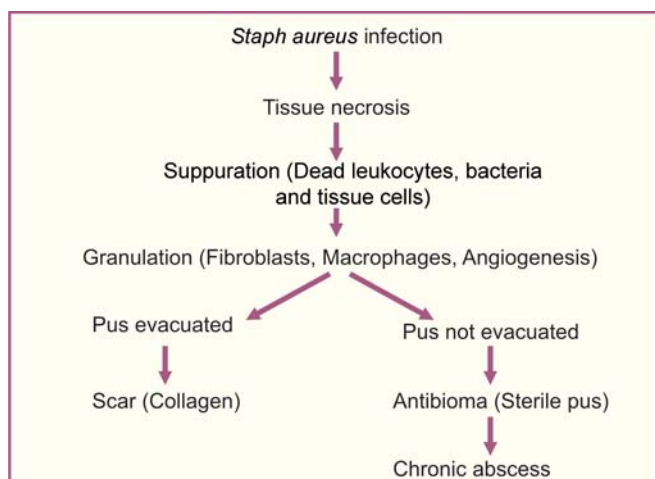
It is a localized collection of pus surrounded by an area of inflammation (Fig. 3.2). It is usually caused by staphylococcal infection. The organisms reach the infected area by following routes:

- Hematogenous route
- Local extension from adjoining area of infection
- From outside (penetrating wounds)

Pathophysiology

There is collection of polymorphonuclear leukocytes at the site of infection, which release proteolytic enzymes. These enzymes cause liquefaction of tissues leading to pus formation. The area around the pus is infiltrated by leukocytes and bacteria and is called pyogenic membrane. As abscess grows, it tracks along the plane of least resistance towards skin. The progress of an abscess is shown in Box 3.7.

Box 3.7: Formation and progress of abscess



Symptoms

Malaise, fever, localized swelling with throbbing pain.

Signs

The five classical signs of inflammation are seen: (1) Heat, (2) Redness, (3) Tenderness, (4) Swelling, and (5) Loss of function.

Fluctuation is a late sign and should not be elicited in an acute abscess because of intense pain and inflammation.

Differential diagnosis of abscess are given in Box 3.8.

Treatment

Once pus formation occurs, it should be surgically drained because penetration of pus by antibiotics is poor.

Incision and drainage of abscess should ideally be done under general anesthesia since it is very painful and local anesthesia is not as effective in areas of acute

Box 3.8: Differential diagnosis of abscess

- Ruptured thrombosed aneurysm
 - Past history of local swelling
 - No throbbing pain
 - Mildly tender
 - Mild fever
- Soft tissue sarcoma
 - Diffuse swelling
 - Dilated veins on surface
 - Local temperature raised
 - Non-tender
 - No fever
- Cellulitis

inflammation. A liberal stab incision is made on the most prominent part of the abscess and pus is drained. The fibrous loculi within the abscess cavity are broken with blunt dissection (finger or artery forceps) to make it a single cavity. It helps in better drainage of pus. The cavity is irrigated with antiseptic solution. The granulation tissue in the wall of abscess cavity bleeds profusely. The bleeding is controlled by packing the abscess cavity with a roller gauge soaked in antiseptic solution (povidone iodine) and wound is dressed. The pack is removed after 48 hrs and bleeding stops by that time. After that daily dressing is done with antiseptic solution and the cavity is lightly packed with gauze till the abscess heals. This gauze prevents early closure of skin wound and the wound cavity heals from the floor and thus abscess does not recur. Due to development of MRSA strain (Methicillin Resistant *Staph aureus*), amoxycillin with clavulanic acid is given in dosage of 1 gm BID for 5-7 days. However, antibiotics can be changed according to report of pus culture and sensitivity.

Hilton's method of incision and drainage should be used in 'high-risk' anatomical areas (Box 3.9). Areas like neck, axilla and groin have vital vessels and nerves, which are likely to be injured during abscess drainage. Hence, skin and subcutaneous tissue (only) are incised

Box 3.9: 'High-risk' anatomical areas of abscess

Site	Structures at risk
Neck	Carotid and subclavian vessels
Axilla	Axillary vessels
Groin	Femoral vessels
Parotid region	Facial nerve



Fig. 3.3: Abscess axilla. Beware of ruptured aneurysm!
Always aspirate before incising

with stab knife and abscess cavity is then opened by gently thrusting a pair of sinus forceps into the abscess cavity.

Cautions in abscess drainage: A ruptured thrombosed aneurysm has local signs of inflammation and mimics an abscess. If it is drained by mistake, it can cause fatal hemorrhage. Hence, if an abscess is located in a high-risk area, presence of pus should always be confirmed by needle aspiration before attempting surgical drainage (Fig. 3.3).

Deep-seated abscess is usually difficult to diagnose since classical signs of inflammation are missing. It is best localized by radiological imaging (USG, CT or MRI) and aspirated with a wide bore needle.

ANTIBIOMA

If pus is not drained and abscess is treated with prolonged antibiotics, it leads to formation of sterile pus surrounded by thick fibrous tissue. It makes a hard lump which becomes clinically difficult to differentiate from malignancy, e.g. breast antibioma mimics carcinoma breast.

CARBUNCLE

“When boil ends, a carbuncle begins”. Carbuncle is a multilocular extension of a boil into the subcutaneous tissue.

It is caused by *Staphylococcus aureus* infection. It is usually seen in males after the age of 40 years who have

Box 3.10: Outlines of Carbuncle ‘C’

Definition	Cutaneous and subcutaneous infective gangrene
Etiology	Cocci
Pathology	Communicating abscess
Clinical features	Central necrosis Cribiform appearance
Treatment	Control diabetes Clavulinic acid with amoxycillin Cruciate incision and wound debridement

underlying diabetes mellitus. The common sites are nape of neck and dorsum of trunk (Box 3.10).

Clinical Features

The patient complains of diffuse painful swelling; and within a few days overlying skin becomes necrosed and starts discharging pus. Multiple small necrotic skin areas develop around central necrotic area and these all join to form large area of ulceration (Fig. 3.4).

Treatment

- General measures to improve health and control of diabetes.
- Amoxycillin with clavulinic acid is given and antibiotics may be changed according to pus culture and sensitivity.
- During initial stage, local antiseptic cleaning and osmotic paste (glycerine with magnesium sulphate) may abort the carbuncle and it may heal without skin ulceration.



Fig. 3.4: Carbuncle



Fig. 3.5: Cellulitis leg

- Once skin ulceration occurs, it requires debridement and regular wound dressing.
- Small wounds will contract and heal with scarring while large wounds may require skin grafting.

CELLULITIS

It is the nonsuppurative inflammation of subcutaneous tissues. It is usually caused by hemolytic streptococci or staphylococci which gain entry into the tissues through a scratch, abrasion or surgical wound.

In a child having cellulitis without any skin breach, always think of underlying bone infection.

Clinical Features

There is widespread swelling, redness and pain without definite localization (Fig. 3.5). Soon the skin becomes shiny and boggy especially in areas having loose skin (face, scrotum). To differentiate it from abscess (Box 3.11), the cellulitis is said to have:

No edge No limit
No pus No fluctuation

In untreated and neglected cases, cellulitis may progress to abscess formation, skin necrosis and even septicemia.

Box 3.11: Abscess vs cellulitis

Abscess	Cellulitis
Well circumscribed	Diffuse
Limit is defined	No limit
Contains pus	No pus
Fluctuant	Nonfluctuant

Treatment

- Bed rest and elevation of the part to reduce edema.
- Local application of osmotic paste of glycerin with magnesium sulphate is hygroscopic and reduces edema.
- Injection crystalline penicillin 10 lac units, intravenous, 6 hourly after sensitivity test for five days is useful in spreading streptococcal infection.
- Amoxycillin with clavulanic acid 1 gm. twice a day for 5 days (oral or injectable) is effective for staphylococcal infection.
- Analgesics and anti-inflammatory drugs for control of pain and inflammation.

CELLULITIS IN SPECIAL SITES

Orbit

Infection spreads from paranasal sinuses and causes orbital cellulitis. There is:

- Proptosis (bulging eyeballs)
- Chemosis (conjunctival edema)
- Ophthalmoplegia (impaired ocular movements)
- Diminished vision due to pressure on optic nerve.

Uncontrolled infection may have intracranial extension leading to meningitis and cavernous sinus thrombosis.

Early detection of this condition and prompt use of antibiotics can help in prevention of these complications.

Neck

Cellulitis of submental and submandibular region occurring beneath deep cervical fascia is called as **Ludwig's angina** ("Angina" means to "throttle").

The infection is caused by virulent streptococcal infection along with anaerobes. The precipitating factors are 4 'C':

- Caries teeth
- Carcinoma oral cavity
- Chronic sialadenitis (involving submandibular gland)
- Chemotherapy

Clinical Features

- There is brawny swelling of submandibular region along with inflammatory edema of mouth (Fig. 3.6).

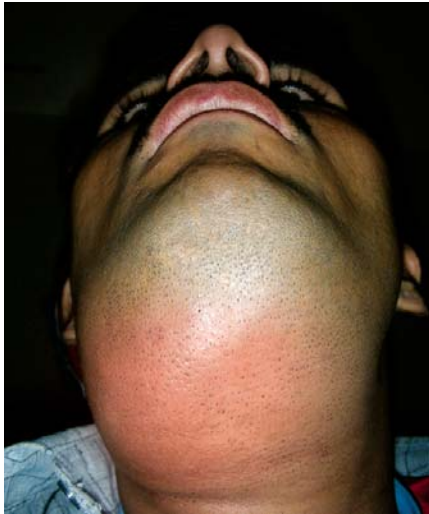


Fig. 3.6: Ludwig's angina

- The patient looks toxic, is febrile and always has putrid halitosis (foul smelling breath).
- Edema of floor of mouth displaces the tongue upwards and backwards causing dysphagia.
- In untreated cases, patient may have laryngeal edema presenting as stridor and choking (Box 3.12).

Box 3.12: Complications of cellulitis in neck

- Laryngeal edema
- Stridor
- Mediastinitis
- Septicemia

Treatment

- Hospitalization and early use of parenteral antibiotics (cefuroxime, amoxycillin with clavulinic acid) and metronidazole for anaerobes.
- If patient does not improve with conservative treatment, surgical drainage should be done.
- Under GA or LA, a curved incision is given below the mandible to incise deep cervical fascia liberally. The mylohyoid muscle may also be incised to decompress the floor of mouth. Wound is irrigated and sutured loosely over subcutaneous drain.
- Rarely tracheostomy may be required in cases of laryngeal obstruction.

LYMPHANGITIS

It is the inflammation of lymphatic pathways that presents as painful red streaks in the skin. It is usually

caused by hemolytic streptococcal infection. It is mostly accompanied by inflammatory enlargement of draining lymph nodes, which are painful and tender (lymphadenitis). In case of infection of hand or fingers red streaks are seen in forearm and axillary lymph nodes are enlarged and tender.

Treatment is antibiotic therapy and rest.

ERYSIPELAS

It means "Red Skin" in Greek.

It is acutely spreading inflammation of skin and subcutaneous tissue with associated lymphangitis.

It is usually caused by hemolytic streptococcal infection. The precipitating factors are malnutrition, poor hygiene and extremes of ages. The lesion develops around a skin abrasion and spreads rapidly as a 'rose pink' rash. The skin is red, swollen and tender and there is distinct line of demarcation at the advancing margin of infection. It commonly involves the face affecting nose and cheeks in a "butterfly lesion". The patient has systemic features in form of fever, chills and prostration. A brownish discoloration of skin remains once the rash fades away.

Erysipelas of face is sometimes difficult to distinguish from cellulitis. However, this distinction is of only academic interest since treatment remains the same, i.e. antibiotics. **Milian's ear sign** helps in distinguishing the two conditions. As facial erysipelas spreads, it involves the pinna as well due to cutaneous lymphangitis. But cellulitis stops short of the pinna since it is inflammation of subcutaneous tissue and in the region of pinna; skin is closely adherent to the cartilage.

Treatment

Injection crystalline penicillin 10 lac units, intravenous, 6 hourly is given for 7-10 days along with local antiseptic application.

BACTEREMIA

It is defined as bacteria circulating in the blood without toxins or clinical manifestations (Box 3.13). It is usually transient and may last for a few minutes since body defenses destroy these organisms. It may follow dental procedures, debridement of infected wounds, etc. It can be dangerous when patient has prosthetic implant since the implant can get infected. Hence, a surgical procedure should be done under cover of antibiotics.

Box 3.13: Definitions

- **Bacteremia** – Bacteria in blood.
- **Septicemia** – Bacteria + Toxins in blood.
- **Toxemia** – Toxins (only) in blood.
- **Pyemia** – Bacteria + Toxins in blood leading to multiple abscesses in the body.

SEPTICEMIA

It is defined as bacteria as well as their toxins circulating in the blood (Box 3.13). It has systemic manifestation in form of fever, rigors, chills, tachycardia and hypotension.

It is caused by streptococci, staphylococci and gram negative bacilli. The organisms enter the circulation when procedures are performed in infected tissues (e.g. tooth extraction in abscess).

Treatment

- Systemic antibiotics, change antibiotics according to blood culture and sensitivity report.
- Hydrocortisone.
- Plasma expanders, blood transfusion.

Prevention

The procedures should be performed under antibiotic cover.

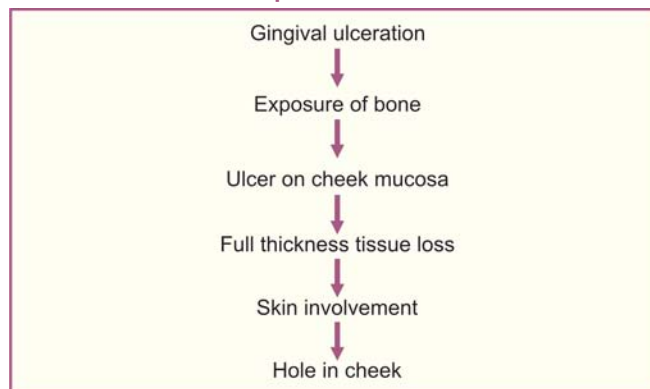
TOXEMIA

Toxins are circulating in the blood without presence of bacteria (producing these toxins) in circulation (Box 3.13). For example, toxins produced by *Clostridium welchii* causing gas gangrene.

PYEMIA

It is septicemia in which bacteria and their toxins are carried in the blood stream and subsequently they produce multiple focal abscesses in different parts of the body (Box 3.13). The features of these abscesses are:

- These are multiple and deep seated.
- Local signs of inflammation (redness, tenderness, pain) are minimal.

Box 3.14: Spread of Cancrum oris

It is usually seen in malnourished children and organism responsible is *Staph aureus*.

Treatment

- Antibiotics.
- General measures to improve nutrition.
- Multiple incisions to drain the abscesses.

CANCNUM ORIS

It is severe ulcerative form of stomatitis affecting malnourished children and spreads rapidly (Box 3.14). Commonest predisposing cause is measles, but it may follow other acute illness as well (typhoid, gastroenteritis). Causative organism is *Borrelia vincentii* that starts as Vincent's stomatitis.

Initially, painful purple papule appears on alveolar margin of the gum. An ulcer then forms exposing underlying bone and extending to cheek or lip which become tender and swollen. In 2-3 days, soft tissue gets sloughed leading to full thickness tissue loss and a hole in cheek or lip. There is foul smelling discharge. The bone and teeth get sequestered. If uncontrolled, child may develop septicemia and die.

Treatment

- IV Penicillin and Metronidazole.
- Regular wound care.
- High protein diet with nasogastric tube feeding.
- Small ulcer may heal with scarring
- Full thickness skin loss may require a pedicle flap at a later date to fill the defect.



4

Chapter

Specific Infections

Sanjay Marwah

TUBERCULOSIS

The infection is caused by acid fast bacillus-*Mycobacterium tuberculosis*.

Modes of Spread

Droplet Infection

A patient of pulmonary tuberculosis on coughing spreads airborne infection. This infection follows three routes:

- Direct spread to lungs through breathing.
- Infection reaches the tonsils and then to cervical lymph nodes.
- Infection reaches the blood and may involve any organ of the body, viz bone, joints, liver, kidneys, brain etc. (**Miliary tuberculosis**).

By Ingestion

In case of bovine tuberculosis, infected milk of a tuberculous cow, if taken without boiling can cause intestinal tuberculosis. It involves ileocecal region and mesenteric lymph nodes. If infected sputum is swallowed by the patient, it can also cause intestinal tuberculosis.

Clinical Features

Pulmonary Tuberculosis

The primary infection is usually asymptomatic and “heals” spontaneously. The disease usually manifests when dormant bacteria are reactivated due to lowered body immunity. The clinical features are: cough, expectoration, hemoptysis (blood in sputum); constitutional symptoms in form of evening rise of temperature, loss of weight and loss of appetite. Management of pulmonary tuberculosis is given in Box 4.3.

Tuberculous Lymphadenitis

It is a common condition in Indian subcontinent mostly affecting cervical lymph nodes. Majority of the patients are children and young adults. The tonsillar (jugulodigastric) lymph node is often the first to become enlarged. It is followed by widespread cervical lymphadenitis as well as involvement of other group of lymph nodes. In untreated cases, the tuberculous lymph nodes pass through following stages:

Stage I Solid enlargement of lymph nodes, which are matted together due to periadenitis.

Stage II The lymph nodes break down due to caseation necrosis and liquid material leaks through the capsule of lymph nodes. Thus **cold abscess** forms and remains confined deep to deep cervical fascia (Box 4.1A and B). If abscess is large, fluctuation may be elicited.

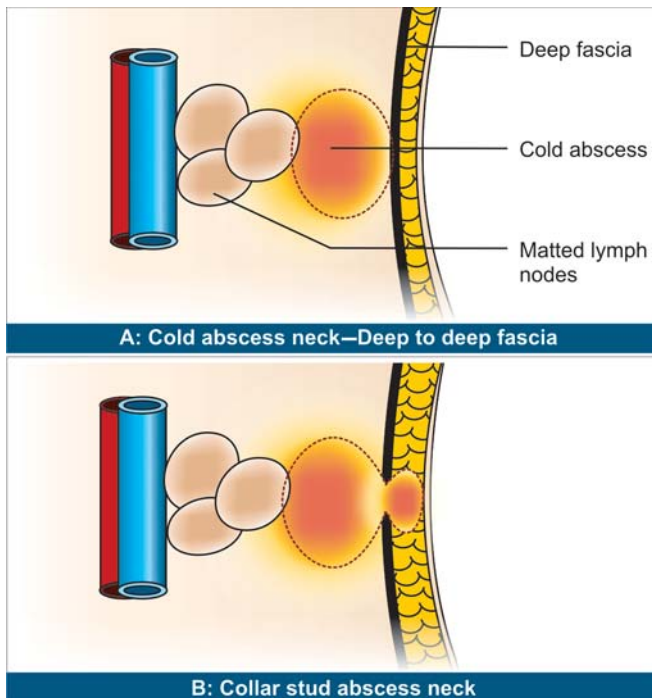
Box 4.1A: Cold abscess

- The term is misnomer since it is not cold to touch.
- Unlike pyogenic abscess (hot to touch), the pus in cold abscess is at body temperature.
- Overlying skin is normal.
- It is nontender.
- Fluctuant.
- Non-transilluminant.
- Cough impulse may be positive (if communicating with body cavity, e.g. pleural cavity).
- Aspiration reveals thin, light yellow, odorless pus.

Stage III After a few weeks, deep cervical fascia is eroded and ‘cold abscess’ enters the subcutaneous plane. It is called as **collar stud** abscess due to its shape (Fig. 4.I). It makes a prominent subcutaneous swelling which is fluctuant. Careful palpation may reveal matted lymph node mass deep to the abscess.

Box 4.1B: Causes of cold abscess

- Tuberculosis
- Maduramycosis
- Actinomycosis
- Leprosy

**Fig. 4.1:** Formation of collar stud abscess**Fig. 4.2:** Multiple discharging tubercular sinuses in neck showing caseation necrosis**Fig. 4.3:** Multiple healed scars of tubercular sinuses

Stage IV If still left untreated, the abscess enlarges and overlying skin becomes reddened. It finally bursts and results in a discharging sinus (Fig. 4.2). If all the pus and necrotic material is expelled, the sinus might heal. Such cases present with multiple scars due to healed sinuses along with matted lymph nodes in the neck (Fig. 4.3).

Intestinal Tuberculosis

It presents as subacute intestinal obstruction, mesenteric lymphadenitis or tubercular ascites.

Miliary Tuberculosis

The infection in blood can spread to involve any organ of the body.

Tuberculosis of Bone and Joint

The disease involves intra-articular bone and synovial membrane respectively. The involvement of spine by tuberculosis is called **Pott's Disease** or **Caries spine**. It is most commonly seen in thoracolumbar region. There is involvement of adjoining vertebrae leading to their collapse and forward bending of spine (kyphosis). Compression of spinal cord may cause paraplegia. Cold abscess forms in paravertebral region and may produce discharging sinus in groin or back.

Lupus Vulgaris

It is cutaneous tuberculosis mostly affecting face. One or more cutaneous nodules appear and there is congestion of surrounding skin. On pressing the lesion

with a glass slide, surrounding hyperemia disappears and 'apple jelly' like nodules becomes apparent. Gradually skin over nodules ulcerates and healing occurs with fibrosis. The ulcer heals at the center and remains active at the periphery and gradually spreads like a wolf (Lupus means wolf). Extensive fibrosis of facial skin gives appearance of 'leonine facies'. Lymphatic obstruction may lead to edema of face. It is premalignant and squamous cell carcinoma may develop in a lupus scar.

Head and neck manifestations of tuberculosis are given in Box 4.2.

Box 4.2: Head and neck manifestations of tuberculosis

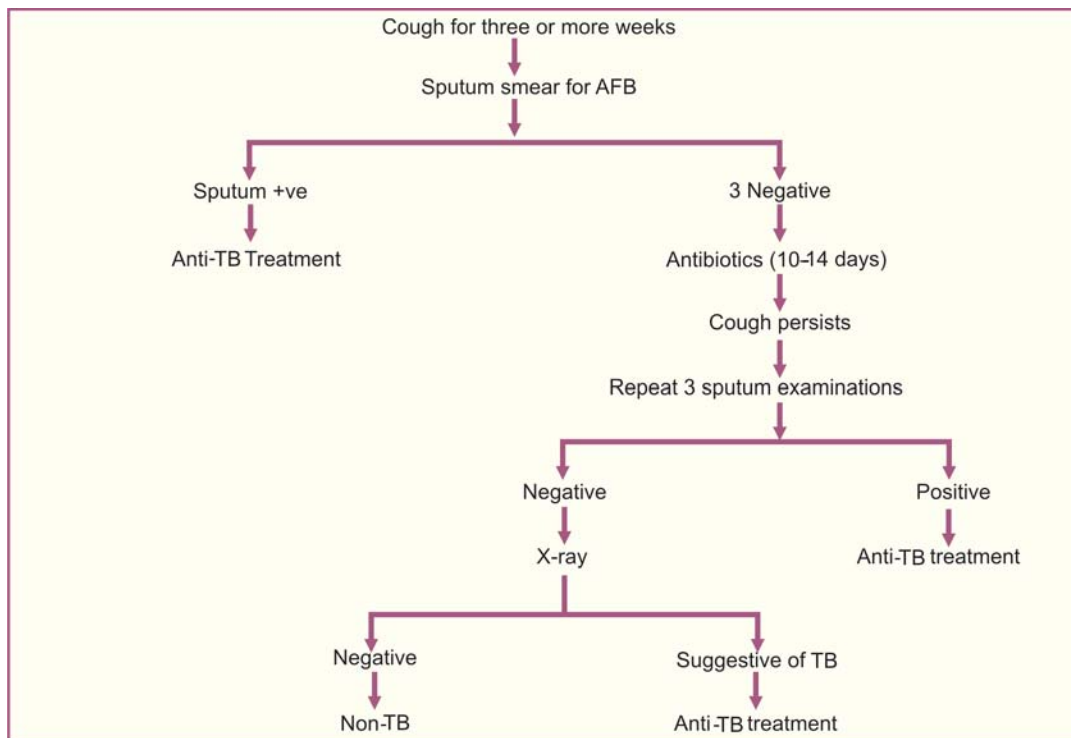
- Cervical lymphadenopathy (matted).
- Cold abscess.
- Non-healing sinus.
- Lupus vulgaris
- Caries cervical spine.

Investigations

- Complete hemogram shows anemia and lymphocytosis.

- ESR is raised.
- Mantoux intradermal test is positive.
- ELISA test is a serological test for tuberculosis and may be positive.
- Sputum examination may show gram-positive tubercular bacilli.
- Chest X-ray may show fibrocavitary lesion (usually in right upper lobe), calcification, pleural effusion.
- FNAC of enlarged cervical lymph node shows tuberculosis in >90% cases.
- Lymph node biopsy shows caseating granulomas. There is area of central caseation surrounded by epithelioid cells, Langhans' giant cells and lymphocytes.
- Aspiration of cold abscess—it may be positive for acid fast bacilli. However, the aspirated pus may be tested for PCR (polymerase chain reaction). It is highly sensitive test since it can pick up even few DNA strands of tubercular bacilli.
- Wedge biopsy of tubercular sinus might show caseating granulomas.
- Pus culture and sensitivity—growth of bacteria takes six weeks and they are seen with Ziehl-Neelsen stain. By 'Bactac method' positive culture can be obtained in two weeks time.

Box 4.3: Management algorithm for pulmonary tuberculosis



Treatment

- Mainstay of treatment is antitubercular chemotherapy (Box 4.4).
- Combination chemotherapy using multiple drugs is given for six months to treat the infection and to prevent the development of resistance.
- Genitourinary and bone tuberculosis requires treatment for 9 months to 1 year.
- Four drugs—INH, Rifampicin, Pyrazinamide and Ethambutol (H,R,Z,E) are given for two months followed by two drugs—INH and Rifampicin (H,R) for another four months.
- The dosage are as follows:-
 - INH 300 mg/day (6 mg/kg)
 - Rifampicin 450-600 mg/day (10 mg/kg)
 - Pyrazinamide 1500 mg/day (30 mg/kg)
 - Ethambutol 800 mg/day (25 mg/kg)

Box 4.4: Names of antitubercular drugs

Isoniazid
Rifampicin
Pyrazinamide
Ethambutol
Streptomycin
Thiacetazone
Kanamycin and amikacin
Capreomycin
Ethionamide and prothionamide
Fluoroquinolones
Cycloserine
P-aminosalicylic acid

Side Effects (Box 4.5)

- INH causes peripheral neuritis. Hence, tablet pyridoxine 10 mg OD should be given along with INH.
- Rifampicin is hepatotoxic. It also causes reddish discoloration of urine and body fluids(saliva, sweat etc.)
- Pyrazinamide is also hepatotoxic. It should be avoided in patients of gout.
- Ethambutol causes visual impairment due to retrobulbar neuritis.

Other Measures

High protein diet and vitamin supplementation.

Multiple Drug Resistance (MDR) Tuberculosis

Causes

- Inadequate treatment or noncompliance of drug treatment
- Infection in immuno-compromised patients, e.g. AIDS patients.
- Infection with atypical mycobacteria, e.g. *Mycobacterium kansasii*, *M. avium*, *M. fortuitum*.

Such cases show no response with routine antitubercular chemotherapy. Culture of tubercular material (e.g. cold abscess) is required for identification of specific species and the treatment is given according to drug sensitivity report. The treatment may last for 1-1½ years. The second line chemotherapy for such

Box 4.5: Side effects of antitubercular drugs

Symptoms	Drugs	Action to be taken
GI upset	Any of the drug	• Reassure • Give drugs over a prolonged period of time (e.g. 20 minutes) • Antiemetics
Itching	INH (other drugs also)	• Reassure • If severe, stop all drugs and re-evaluate
Burning in hands and feet	INH	• Pyridoxine 10 mg/day
Joint pain	Pyrazinamide	• If severe, stop Pyrazinamide
Impaired vision	Ethambutol	• Stop Ethambutol
Loss of hearing, ringing in ears, dizziness	Streptomycin	• Stop Streptomycin
Jaundice	INH, Rifampicin, Pyrazinamide	• Stop all three drugs

cases include: Ciprofloxacin, Ofloxacin, Ethionamide, Capreomycin, Cycloserine.

Directly Observed Treatment (DOT) for Tuberculosis

The failure to take medication as prescribed is a universal phenomenon especially in chronic diseases. This is responsible for development of multi-drug resistant tuberculosis. In view of this, revised national tuberculosis control program has incorporated the concept of “Direct Observation of Treatment (DOT)”. It means that every dose is administered under direct observation. The treatment observer ensures that medicines are taken at correct intervals and in correct dosage. It has the benefit of high cure rate and dramatic reduction in the development of drug resistance. Also, adverse effects are quickly identified and treated accordingly.

D	O	T	S
Directly	Observed	Treatment	Short course
Dedicated	Organized	Team of	Sincere TB workers

Once treatment is given under direct observation, then short course (6 months) of intermittent chemotherapy (thrice a week) is found to be equally effective. Dosage and treatment schedule of intermittent chemotherapy is given in Box 4.6 and Box 4.7 respectively.

Treatment of Cold Abscess

- Small cold abscess mostly resolves with anti-tubercular chemotherapy.
- Large sized cold abscess not responding to drugs needs aspiration. The aspiration should be done with

Box 4.6: Adult dosage of antitubercular drugs for thrice weekly regimen

INH	600 mg
Rifampicin	450 mg (Patients more than 60 kg are given 600 mg of Rifampicin)
Pyrazinamide	1500 mg
Ethambutol	1200 mg
Streptomycin	0.75 gm (patients more than 50 years of age and weighing less than 30 kg are given 0.5 gm of Streptomycin)

a wide bore needle (18-20 G) and it should be antigravity or nondependent aspiration so as to prevent formation of persistent sinus along the needle track. Sometimes, repeated aspirations are required at interval of 1-2 weeks.

Surgical Treatment

If there is a persistent cervical sinus with underlying lymph node mass which fails to resolve with drug treatment, it needs surgical excision. Due to periadenitis and fibrosis, the lymph nodes in neck may be adherent to adjoining internal jugular vein, carotid artery and vagus nerve. Hence, liberal incision and wide exposure under general anesthesia is necessary for dissecting lymph node mass from these vital structures. The excised tissue should be subjected to histopathology and culture sensitivity.

In case of caries spine, immobilization of spine is done to prevent spinal cord damage using a plaster jacket (Minerva jacket). Unstable spine requires operative fixation by spinal fusion operation (arthrodysis).

SYPHILIS

It is a sexually transmitted infection caused by *Treponema pallidum*, a spirochaete.

Box 4.7: Treatment schedule for tuberculosis in thrice weekly regimen

Category	Type of patient	Treatment
Category-I	New sputum positive case	HRZE × 2 months HR × 4 months
Category-II	Treatment failure/relapse/defaulters	HRZES × 2 months HRZE × 1 month HRE × 5 months
Category-III	Sputum negative or extrapulmonary tuberculosis (e.g., lymph nodes, intestines)	HRZ × 2 months HR × 4 months
H (INH), R (Rifampicin), Z (Pyrazinamide), E (Ethambutol), S (Streptomycin).		

The incidence of syphilis has dramatically reduced all over the world after introduction of penicillin.

The causative organism is spiral shaped and delicate. It dies rapidly on drying. Therefore, infective lesions are mostly seen in moist areas (genitalia, mouth and anus). The organism is able to penetrate skin and mucosa at the point of contact. It causes inflammatory reaction in perivascular lymphatics leading to obliterative endarteritis. Hence, syphilis is essentially a vascular disease.

The organism is present in the lesions only in early syphilis (primary and secondary) and up to 2 years of latent syphilis. So syphilis is infective only during this period. The lesions of tertiary syphilis are not infective since they don't have any organisms in them.

The disease is divided into 4 stages:

Primary Syphilis

- The lesion appears in genitalia after about one month of sexual contact.
- It is a painless, shallow indurated ulcer that feels like a button—called as **Hunterian chancre**.
- The draining lymph nodes are enlarged, nontender, discrete and rubbery in consistency.
- The extragenital chancre can develop on lip, tongue and nipple.

Diagnosis

- Dark field examination of smear prepared from the lesion shows highly motile, spiral shaped organism—*Treponema pallidum*.
- Serological tests—these tests become positive after one month of appearance of chancre.
- Non-specific tests—these can be positive in non-treponemal conditions as well (malaria, glandular fever, etc.). These are VDRL, Wassermann and Kahn test.
- Specific tests:
 - ☐ TPI—*T. pallidum* immobilization test.
 - ☐ TPHA—*T. pallidum* hemagglutination test.
 - ☐ FTAB—Fluorescent treponema antibody test.
 - ☐ CFT—Complement fixation test.

Secondary Syphilis

- The signs appear in 1½- 3 months of infection.
- Generalized skin rash (reddish color).

- Small superficial erosions in mouth which join together to form **Snail track ulcers**.
- Fleshy wart like lesions on genitalia (**condyloma lata**).
- Generalized lymphadenopathy.
- Sore throat, 'moth eaten' alopecia, iritis, bone and joint pains.
- Constitutional symptoms like fever, malaise and headache.

Latent Syphilis

Untreated secondary syphilis changes into latent syphilis that lasts from 2 years to lifetime. Although serological tests are positive, but there are no clinical signs.

Tertiary Syphilis

The typical lesion in this stage is **gumma** that forms due to hypersensitivity reaction. It consists of granulation tissue with central necrosis. It usually starts as a subcutaneous swelling that tends to occupy midline of the body (posterior 1/3rd of tongue, sternoclavicular joint). Soon central softening occurs and a characteristic ulcer forms with following features:

- Painless ulcer
- Punched out margins
- 'Wash leather' floor
- Heals with thin scarring

In 5-15 years time, patients develop

- Neurosyphilis
- Cardiovascular syphilis

Head and neck manifestations of acquired syphilis are given in Box 4.8.

Box 4.8: Head and neck manifestations of acquired syphilis

Primary syphilis:

- Chancre of tongue, lip.
- Discrete, 'shotty', cervical lymph nodes.

Secondary syphilis:

- Sore throat
- Hoarseness
- 'Moth eaten' alopecia
- Iritis
- 'Snail track ulcers' in oral cavity
- Cervical lymphadenopathy

Tertiary syphilis:

- Gumma (posterior 1/3rd of tongue, sternum)
- Neurosyphilis

Congenital Syphilis

It is caused by *T. pallidum* crossing the placenta from an infected expectant mother to the fetus. The fetal infection leads to intrauterine death or death in early infancy.

Early Congenital Syphilis

The signs seen in a newborn are in form of:

- Rhinitis and nasal discharge
- Hepatosplenomegaly
- Epiphysitis
- Osteochondritis

Late Congenital Syphilis

The signs are seen during childhood or puberty in form of **Hutchinson's Triad**:

1. Interstitial Keratitis—bilateral corneal haziness
2. 8th nerve deafness
3. Hutchinson's teeth—peg-shaped deformity of upper central incisors.

Other signs include: Saddle nose, palatal perforation, parietal bossing, Clutton's joints.

Head and neck manifestations of congenital syphilis are given in Box 4.9.

Box 4.9: Head and neck manifestations of congenital syphilis

Early:

- 'Snuffles' (rhinitis, nasal discharge)

Late:

- Interstitial keratitis
 - 8th nerve deafness
 - Hutchinson's teeth
 - Saddle nose
 - Palatal perforation
 - Parietal bossing
- Hutchinson's triad

Treatment

- Injection procaine penicillin 6 lacs units I/M OD is given for 15 days in primary and secondary syphilis. In tertiary syphilis, it is given for 21-30 days.
- For penicillin sensitive patients, tetracycline, erythromycin or cephalexin is given.
- **Jarisch Herxheimer Reaction**—after 6 hrs of first injection of penicillin, half the cases of early syphilis

develop fever with rigors and chills that lasts for a few hours.

- Congenital syphilis can be prevented as well as treated by giving 6 lac units of P. Penicillin to the expectant mother for 15 days.

GONORRHEA

- Sexually transmitted disease with incubation period of 4-7 days.
- It is caused by *Neisseria gonorrhoeae*, a gram negative, kidney-shaped diplococcus.
- It infects:
 - Anterior urethra in males.
 - Urethra and cervix in females.
 - Oropharynx, rectum and anal canal in both sexes.
- The main symptom is urethral discharge with burning micturition.

Diagnosis

- Gram staining of urethral smears show gram-negative diplococci.
- Two glass urine test—there is haziness in first glass and urine is clear in second glass. It shows that pus is passed in first part of urine.

Treatment

Procaine penicillin or ciprofloxacin are the antibiotics of choice. Probenecid is added to increase the effective concentration of antibiotic by delaying its excretion. In cases that are resistant or allergic to penicillin, Kanamycin is used as second line drug.

Complications

Local

- Epididymo-orchitis
- Periurethral abscess
- Urethral stricture
- Prostatitis
- Proctitis
- Salpingitis in females

Systemic

- Arthritis
- Iridocyclitis
- Endocarditis

Ophthalmia neonatorum Although rare now, gonococcal infection used to be an important cause of blindness in newborn.

ANTHRAX

It is caused by *Bacillus anthracis*, a gram positive, spore forming and aerobic rod. The disease is primarily seen in cattle but human beings can be affected while handling animal hides, carcasses and wool.

The lesion mostly involves skin of exposed parts of the body (face, hands, forearms). It starts as an itching indurated papule. Soon it is replaced by black central scab surrounded by a ring of vesicles and this lesion is called as **malignant pustule**.

- Pus and pain are absent in the lesion.
- Regional lymph nodes are enlarged (Box 4.10).
- Toxemia may occur.

Diagnosis

Smear of vesicle fluid shows gram-positive rods.

Differential Diagnosis

Severe furuncle (Box 4.11).

Treatment

Penicillin is the drug of choice.

Rarely anthrax may involve:

- Lungs due to inhalation of spores.
- Intestines due to ingestion of spores.

Box 4.10: Head and neck manifestations—Anthrax

- 'Malignant pustule' on face
- Cervical lymphadenopathy

Box 4.11: Differences between Furuncle and Anthrax

Furuncle	Anthrax
• Caused by <i>Staph aureus</i> • Abscess of sweat glands • Throbbing pain and swelling • Small pustule forms, ruptures and discharges pus • Treatment—Cloxacillin	• <i>Bacillus anthracis</i> • Skin involvement by Anthrax • Itching indurated papule, no pain • Small black scab surrounded by ring of vesicles, no pus • Treatment—Penicillin

ACTINOMYCOSIS

The disease is caused by *Actinomyces israelii*. It is a gram-positive, anaerobic, branching, filamentous organism, also known as 'ray fungus'.

It is present in normal oral flora and invades the tissues in presence of carious teeth or following trauma.

In the tissues, the organism causes sub-acute pyogenic inflammation. There is formation of abscess, which is surrounded by connective tissue and granulation tissue. The abscess gradually expands into adjoining tissues and forms burrowing, tortuous sinuses that burst outside and discharge pus.

Clinical Features

There are four clinical types of actinomycosis:

Facio-cervical

- Commonest type.
- Lower jaw is mostly affected adjacent to a carious tooth.
- The gum becomes indurated and overlying skin becomes nodular. The abscesses burst through the skin. Multiple indurated sinuses appear on lower jaw and neck.
- Cervical lymph nodes are not enlarged.
- *Differential diagnosis*: Carcinoma floor of mouth, jaw tumor, chronic osteomyelitis of mandible.
- Head and neck manifestations of actinomycosis are given in Box 4.12.

Thoracic

- It reaches lungs by inhalation of organism.
- Initially lungs are involved followed by pleura and then chest wall. Multiple discharging sinuses are seen on chest wall.

Right Iliac Fossa

- Following appendicectomy, the organism invades paracecal tissue and produces an indurated mass in right iliac fossa.
- It does not compromise the bowel lumen. Later, multiple discharging sinuses appear in right iliac fossa.

Liver

- The organism from right iliac fossa may reach liver via portal vein.

- The liver tissue is gradually destroyed and replaced by multiple abscesses (**Honeycomb liver**).

Diagnosis

- The discharge is collected in a test tube and inspected against good light. It shows pinhead size, shiny sulphur granules.
- Tissue microscopy shows gram-positive branching filamentous organisms.

Treatment

- Prolonged antibiotic course is required to eradicate this low-grade chronic infection.
- The organism is sensitive to penicillin, tetracycline and lincomycin.
- Injection C penicillin 10 lac units once a day is given for 6-12 months.
- The abscesses require drainage with excision of sinuses and resection of damaged tissues.

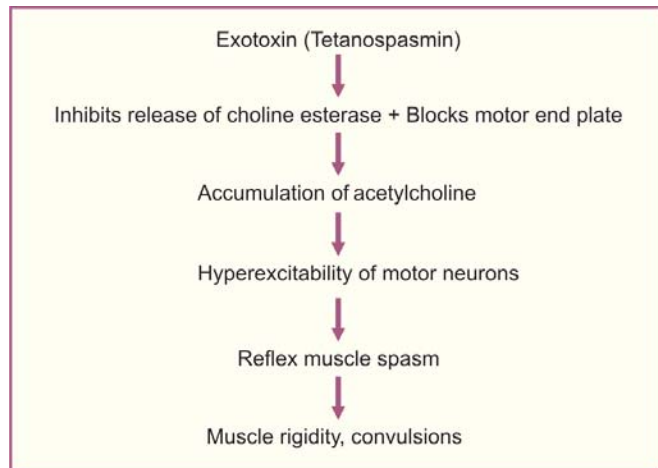
Box 4.12: Head and neck manifestations of actinomycosis

- Involvement of lower jaw (carious teeth present)
- Hypertrophic, indurated gums
- Nodules on facial skin adherent to mandible
- Sinuses and abscesses discharging sulphur granules
- Cervical lymph nodes not enlarged

TETANUS

- The disease is caused by *Clostridium tetani*, a gram-positive rod with a terminal spore (drum stick appearance).
- The organism exists in soil and human intestines.
- The organism itself is noninvasive and enters through site of trauma. It produces neurotoxin that is responsible for symptoms of tetanus.
- A low oxygen tension is required for organism to grow. Thus, it multiplies in presence of dead and necrotic tissue and produces exotoxin.
- The exotoxin locally inhibits the cholinesterase at motor end plate leading to excess of acetylcholine that causes sustained state of tonic muscle spasm in the region of trauma.
- The exotoxin then travels along the nerves and reaches central nervous system and gets fixed there. By the same mechanism, it produces hyperexcitability of motor neurons and reflex muscle

Box 4.13A: Mechanism of action of exotoxin



spasm all over the body. Even minor sensory stimuli like noise can precipitate severe muscle spasm.

- Once fixed to nervous tissue, the exotoxin can not be neutralized leading to irreversible damage (Box 4.13A).
- Common modes of infection are:**
 - ☐ *Wounds* caused by thorns, nails, splinters and road side accidents.
 - ☐ *Umbilical sepsis*: In rural India, umbilical cord of newborn is sometimes cut with rusted instruments and there is a ritual of applying cow dung on the umbilical stump. It can lead to 'tetanus neonatorum'.
 - ☐ *Puerperal tetanus*: It is due to unsterile instrumentation of genital track during delivery.
 - ☐ *Postoperative tetanus*: It is due to break down of sterile technique during surgery or due to wound contamination from patient's own intestinal tract.

No wound → No tetanus

Clinical Features

The average incubation period is 7-10 days. The first symptom is **trismus** (lock jaw*). It is followed by reflex spasm of the muscles. The time interval between the first symptom (trismus) and onset of reflex spasm is called as 'period of onset'. Shorter the 'period of onset', poorer is the prognosis. If 'period of onset' is less than 48 hrs, death is likely. Other features are:

- Dysphagia
- Pain and stiffness in neck, back and abdomen
- Risus sardonicus**—painful smiling appearance due to spasm of facial muscles.

*Trismus can sometimes occur in other head and neck condition as well viz, parotid abscess, alveolar abscess.

- Generalized convulsions—clenched teeth, arched back, extended limbs and tonic contraction of all the body muscles (**opisthotonus**—body bent like a bow).
- Severe spasm may stop respiration and can cause death due to asphyxia. Aspiration pneumonia is another common cause of death.
- Some less common manifestations of disease are:
 - *Local tetanus*: Local contraction of muscles in the neighborhood of wound.
 - *Cephalic tetanus*: It is a type of local tetanus that follows wounds of head and face, otitis media. The symptoms first appear on side of injury. There is irritation or paralysis of cranial nerves.
 - The facial nerve is most commonly affected leading to deviation of angle of mouth.
 - Ophthalmoplegia due to involvement of ocular nerves.
 - Tongue deviation due to involvement of hypoglossal nerve.
 - Trismus and dysphagia may also occur.
 - *Latent tetanus*: The manifestations appear after few months or even years after injury that might have been forgotten.
- Head and neck manifestations of tetanus are given in Box 4.13B.

Box 4.13B: Head and neck manifestations of tetanus

- Lock jaw
- Dysphagia
- Neck stiffness
- Risus sardonius
- Facial palsy
- Ophthalmoplegia ——— Cephalic tetanus
- Tongue deviation

- Differential diagnosis of tetanus is given in Box 4.13C.

Box 4.13C: Tetanus: Differential diagnosis

- Acute tonsillitis
- Acute pharyngitis
- Alveolar abscess
- Acute parotitis
- Arthritis of temporomandibular joint
- Epilepsy
- Meningitis
- Anxiety neurosis

Treatment

- Hospitalization and isolation for providing quiet environment and comfort.
- Surgical care of infected wound by cleaning and debridement. The wound should be left open and dressed regularly.
- Injection C Penicillin (10 lac units 6 hourly) is antibiotic of choice. In case of penicillin sensitivity, tetracycline can be used.
- Injection tetanus toxoid 0.5 ml I/M starts giving active immunization by producing antibodies after about one month.
- Injection human anti-tetanus globulin (250- 500 units I/M) gives passive immunization for about one month. After that antibodies produced by active immunization take over.
- The patients with spasm and convulsions require sedation and muscle relaxants. The drugs used are-
 - Diazepam: 10 mg 6 hrly.
 - Phenobarbitone: 60 mg 6 hrly.
 - Chlorpromazine: 75 mg 6 hrly.
 These drugs are given in rotation in such a way that every 2 hrly patient receives one dose of sedation. The dosage is gradually reduced as patient starts improving.
- If convulsions persist despite sedation, patient is paralyzed with muscle relaxants and put on positive pressure ventilation till improvement occurs.
- Ryle's tube feeding is done to maintain nutrition.
- Removal of visceral stimuli (full bladder, fecal impaction).
- In severe cases, tracheostomy may be needed.
- The patient who has survived tetanus is not immune and unless immunized, he can get second attack of tetanus.

Prophylaxis

- In tetanus prone wounds, if person is previously immunized, booster of tetanus toxoid is given.
- In tetanus prone wounds, if person is not previously immunized, 0.5 ml of tetanus toxoid and 250 units of human anti-tetanus globulin should be given.
- During pregnancy and childhood, immunization schedule should be followed as per WHO guidelines
 - Tetanus toxoid is given twice in first trimester of pregnancy at interval of one month.
 - Tetanus toxoid is given in combination with pertussis and diphtheria vaccines (DPT) at

6 weeks, 10 weeks and 14 weeks of age. A booster is given at 18 months of age. After that, booster is given once in five years.

LEPROSY (HANSEN'S DISEASE)

- Leprosy is a chronic infection caused by the acid fast bacilli (5%) *Mycobacterium leprae*.
- It affects primarily the cooler parts of the body, i.e. skin, upper respiratory tract, anterior segment of eye, superficial portions of peripheral nerves and testes.
- The disease is endemic in areas with hot moist climate and in poor tropical countries.
- Majority of the cases are located in India (78%).
- Commonest route of entry is nasorespiratory tract. Other routes are:
 - Skin to skin transmission (uncommon).
 - Maternofetal transmission across the placenta.
 - Transmission from milk of leprosy patient to infant.
- It is mainly contacted in childhood and late adolescence. Incubation periods are usually 2 to 5 years but vary up to as long as 30 years.

Classification

- There is wide range of clinical and pathological forms of leprosy.
- **Modified Ridley and Jopling classification** divides leprosy into seven groups based on immunologic, pathologic and clinical features (Fig. 4.4):
These are:
 - TT Tuberculoid Polar (High resistance)
 - BT Borderline Tuberculoid
 - TI Tuberculoid Indefinite
 - BB Mid borderline
 - LI Lepromatous Indefinite
 - BL Borderline Lepromatous
 - LL Lepromatous Polar (Low resistance)

In addition, not included in Ridley and Jopling classification, are cases of indeterminate leprosy, pure neural leprosy and histoid leprosy.

Clinical Features

The two main forms of leprosy show distinctive clinical features:

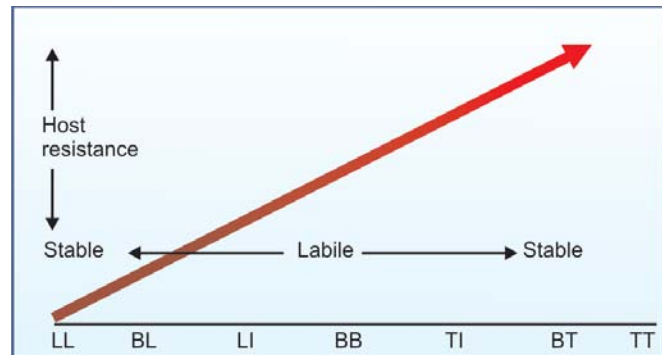


Fig. 4.4: Classification of leprosy

- *Lepromatous leprosy*:
 - Skin lesions are generally symmetrical, multiple, slightly hypopigmented and erythematous macules, papules, nodules or diffuse infiltrates.
 - *Leonine facies* appearance (looks like a lion) due to colascence of nodular lesions and collapse of nose are characteristic features (Box 4.14A).
 - The lesions are hypoesthetic/anesthetic, but sensory disturbances are not as distinct as in tuberculoid forms.

Box 4.14A: Face involvement in leprosy

- Nodular lesions on face (leonine facies)
- Wrinkling of skin (aged look)
- Collapse of nasal bridge due to destruction of cartilage
- Lifting of nasal tip
- Facial nerve palsy
- Incomplete closure of eyes (epiphora, conjunctivitis, keratitis)

- *Tuberculoid leprosy*:
 - The skin lesions occur as either single or as a few asymmetrical lesions which are hypopigmented and erythematous macules.
 - There is distinct sensory impairment.

One of the most characteristic features of leprosy is its effect on nerves. The involved nerves are thickened and tender. The anesthesia that results from nerve involvement is an important point in diagnosis and is also a cause of secondary damage and deformity leading to trophic ulcers and auto-amputation (Box 4.14B).

In males testicular involvement may occur leading to testicular atrophy.

Box 4.14B: Nerve involvement in leprosy

Nerve	Site of involvement	Outcome
Ulnar nerve	Elbow	Claw hand
Median nerve	Wrist	Claw hand
Posterior tibial nerve	Ankle	Claw toe
Lateral popliteal nerve	Below knee	Foot drop
Facial nerve	Bony canal	Facial asymmetry, Exposure keratitis

Box 4.14C: Difference between lepromatous and tuberculoid leprosy

Feature	Lepromatous leprosy	Tuberculoid leprosy
Skin lesion	Symmetrical, multiple, hypopigmented, erythematous, maculopapular or nodular lesions (leonine facies)	Asymmetrical, single or few hypopigmented and erythematous macular lesions
Nerve involvement	Present, but less severe sensory disturbances	Distinct involvement with severe sensory disturbances
Histopathology	Collection of foamy macrophages/ lepra cells in the dermis. Clear zone between epidermis and dermis	Epithelioid cell granulomas with giant cells eroding the epidermis (no clear zone)
Bacteriology	Lepra cells highly positive for lepra bacilli seen as 'Globi' cells or 'cigarettes-in-pack' appearance	Lepra bacilli few in numbers
Immunity	Suppressed (low resistance)	Good immune response (high resistance)
Lepromin test	Negative	Positive

Salient differences between the two main forms are summarized in Box 4.14C.

Diagnosis

- Skin smear shows acid fast bacilli (AFB).
- Skin biopsy shows typical histological features.

Treatment

- A dedicated team of physician, orthopedic surgeon, plastic surgeon and eye surgeon is required for proper treatment.
- Drug treatment includes multiple drug therapy for a prolonged period.
- In Lepromatous (LL) and Borderline Lepromatous (BL) types, three drugs are given for two years.
 - ☐ Dapsone 100 mg/day

- ☐ Clofazimine 50 mg/day
- ☐ Rifampicin 600 mg/month under supervision

At the end of therapy, the skin smear should be negative for AFB.

- In Tuberculoid (TT) and Borderline Tuberculoid (BT) types, two drugs are given for six months.
 - ☐ Dapsone 100 mg/day
 - ☐ Rifampicin 600 mg/month under supervision.
- Role of plastic surgeon is:
 - ☐ Correction of cosmetic deformity of face.
 - ☐ Lateral tarsorrhaphy to prevent exposure keratitis.
 - ☐ Temporalis muscle flap to upper eye lid for prevention of exposure keratitis.
 - ☐ Nasal prosthesis.

- Role of orthopedic surgeon is:
 - Tendon transfer for hands and feet deformities.
 - Amputation.

VIRAL INFECTIONS

- Hepatitis B, Hepatitis C and human immunodeficiency virus (HIV) are of importance to the surgeons since the surgeons can get infected from exposure to these patients and vice versa.
- **Hepatitis** patients give history of jaundice and test positive for hepatitis antigen.
- Hepatitis B vaccine is available and all surgeons and health care workers handling blood and blood products should get vaccinated.
- After exposure, chances of transmitting Hepatitis B infection to the surgeon are much more than transmitting HIV infection.
- **HIV infection** is caused by human immunodeficiency virus type I (HIV-I) that is a retrovirus.
- Blood, semen, vaginal secretions and breast milk can transmit infection. Saliva does not transmit HIV infection.
- High-risk groups for HIV infection are:
 1. Homosexuals.
 2. I/V drug abusers.
 3. Hemophiliacs receiving multiple blood transfusions.
 4. Heterosexual contacts with HIV positive cases.
- After infection, patient gets generalized lymphadenopathy and fever.
- Antibodies to HIV develop 12 weeks after infection and at this stage; diagnosis can be made by serological testing. However, during this 12 weeks period (**window period**), the patient is potentially most infective and yet tests negative for HIV.
- Development of AIDS takes 7-9 years after infection. At this stage, there is functional impairment of CD4+ lymphocytes resulting in disordered antibody production and delayed hypersensitivity reaction. When CD4+ count is less than 200 cells/cmm, it is defined as AIDS.
- Patient with HIV infection may require surgery just like any other routine patient. The usual problems in these cases include—perianal sepsis, lymphoma, Kaposi's sarcoma, peritonitis due to gut perforation and intestinal obstruction (Box 4.15A).
- HIV infection should be suspected in patients presenting with unexplained large abscesses such as shown in Figures 4.5A and B.

Box 4.15A: Usual surgical problems in HIV patients

- Perianal abscess
- Lymphadenopathy (lymphoma, tuberculosis)
- Kaposi's sarcoma
- Intestinal obstruction
- Peritonitis due to gut perforation
- Extensive esophageal ulceration



Fig. 4.5A: Cold abscess of chest wall in HIV positive case



Fig. 4.5B: Large abscess of neck in HIV positive case

- Head and neck manifestations of HIV infection are given in Box 4.15B.
- At time of surgery, if CD4+ count is less than 200 cells/cmm, uncontrolled infection and poor healing is expected after surgery.

Precautions during Surgery in HIV Patients

- HIV infection in health care workers is usually by skin puncture caused by needle stick injury that contains HIV infected blood.
- During surgery, the procedure should be performed in an orderly manner.
- 'Universal precautions' should be used while performing surgery in high-risk group and HIV positive patients (Box 4.15C).

Box 4.15B: Head and neck manifestations of HIV

- Scars of herpes zoster on face and back.
- Pigmented scars of furunculosis.
- Multiple ulcers in oral cavity due to herpes infection.
- Faucial inflammation.
- Thrush.
- Hairy Leukoplakia of tongue.
- Oral Kaposi's sarcoma (purple staining and raised plaque on hard palate).
- Neck abscess (Fig. 4.5B).
- Symmetrical enlargement of posterior cervical, occipital, axillary and epitrochlear lymph nodes.
- Asymmetrical cervical lymph node enlargement due to HIV associated tuberculosis, Kaposi's sarcoma or lymphoma.

Box 4.15C: Universal precautions

- Wear safety spectacles to protect eyes.
- Water proof gown to protect front and arms.
- Full boots to protect feet.
- Wear double pairs of gloves.
- Keep surgical assistants to a minimum.
- Sharp instruments should be passed from scrub nurse to the surgeon in a kidney tray to avoid injury (Fig. 4.6).
- Put used needles in puncture resistant containers and never try to replace them back in protective sheath.
- Health workers with exudative lesions or weeping dermatitis should not handle such patients.
- Wear gloves during procedures (taking blood samples, inserting cannula, dental extractions).



Fig. 4.6: Correct method of passing knife in a kidney tray

Procedure in the Event of Contamination with Infected Blood

- Immediately clean the contaminated area under running water.
- Prophylaxis with zidovudine should be started within one hour of exposure. Dosage is 250 mg BD for one month.
- Prophylaxis of hepatitis should also be given to the surgeon.
- Baseline HIV testing should be done immediately and then repeated at 12 weeks to determine for seroconversion.

Infection of Patient by the Surgeon

- Six patients getting infection during dental procedures by HIV +ve dental surgeons have been reported in literature.
- Hepatitis infection can be transmitted from an infected surgeon to the patient during surgical procedure. The surgeon sustains injury with a sharp instrument and the contaminated instrument then infects the patient due to re-contact.

It is recommended that health care workers who are infected with HIV or hepatitis B should not perform "exposure prone" procedures.

5

Chapter

Sinus, Ulcer and Fistula

Sanjay Marwah

SINUS

It is a blind tract extending from epithelial surface to surrounding tissues. It has one opening. It is lined by granulation tissue or epithelium (Fig. 5.1).

Anatomical Sinuses

These are normally present in the body, e.g. frontal sinus, maxillary sinus.

Congenital Sinus

It is present since birth, e.g. preauricular sinus (Fig. 5.2).

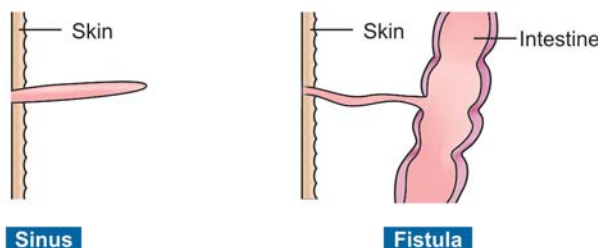


Fig. 5.1: Sinus and fistula

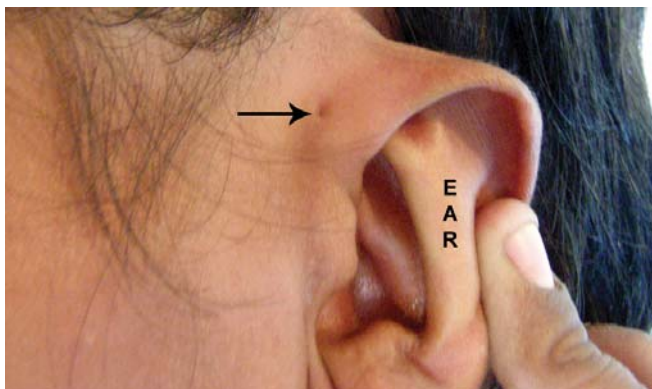


Fig. 5.2: Preauricular sinus



Fig. 5.3: Median mental sinus

Acquired Sinus

Various causes are:

- Tubercular sinus in neck. It occurs following rupture or drainage of cold abscess in the neck. Margins of the sinus are undermined and there is palpable mass of matted lymph nodes.
- Median mental sinus in submental triangle is due to ruptured tooth abscess (Fig. 5.3).
- Pilonidal sinus is a midline sinus in natal cleft. It contains tuft of dead hair with foul smelling discharge.
- Hidradenitis suppurativa. It is abnormality of apocrine glands present in axilla and groin. It presents with recurrent abscesses and multiple discharging sinuses.

FISTULA

It is an abnormal tract between two epithelial surfaces. It has two openings. The tract is lined by granulation tissue or epithelium (Fig. 5.1).

External Fistula

When the tract communicates a hollow viscus (e.g. intestine) to the skin. Examples are: Parotid fistula, thyroglossal fistula, branchial fistula.

Internal Fistula

When the tract communicates with two hollow viscera (e.g. two intestinal lumens, two blood vessels). Examples are: Tracheo-esophageal fistula, oro-maxillary fistula.

Congenital Fistula

It is present since birth. Examples are: Branchial fistula (See Chapter 12), Tracheo-esophageal fistula, Arterio-venous fistula.

Acquired Fistula

Example are:

- Fistula in ano
- Arteriovenous fistula: Following trauma, created surgically for dialysis in renal failure.
- Parotid fistula: Following drainage of parotid abscess.

A sinus or fistula may not heal despite treatment. The causes are given in Box 5.1. These causes need to be identified and removed or treated.

EXAMINATION OF SINUS/FISTULA

History

Present since birth (congenital) or appeared during later life (acquired).



Fig. 5.4: Non-healing sinus cheek following accident

Box 5.1: Causes of non-healing sinus/fistula

- Foreign body in tract (Figs 5.4 and 5.5)
- Non-dependent drainage
- Epithelialization/fibrosis of tract
- Lack of rest to the affected part
- Chronic specific infection (e.g. Tuberculosis)
- Malignancy
- HIV
- Persistent discharge (urine, stool, pus)
- Ischemia
- Malnutrition
- Drugs (steroids, chemotherapy)
- Radiotherapy

CASE SUMMARY

A 20 years male met a road side accident 1 year ago. He sustained multiple lacerations on right side of face that healed within two weeks time. However, a sinus persisted in area of scarring on right cheek (Fig. 5.4). X-ray face showed no abnormality. Biopsy from the ulcer margin was done twice and revealed nonspecific changes. Sinus was explored twice under local anesthesia but recurred. Ultimately patient was hospitalized and the sinus was explored under general anesthesia. To surprise of the surgeon, a piece of wood measuring 3×1.5 cm was delivered through the sinus (Fig. 5.5) and the sinus healed rapidly thereafter.

Learning point: Previous history of trauma to face was ignored in this case that led to delay in diagnosis. This case highlights the importance of history taking.



Fig. 5.5: The same sinus on exploration revealing a piece of wood

Past history of tuberculosis, trauma, drainage of an abscess (cold abscess).

Examination

Site: Specific location is often diagnostic, e.g.

- Parotid fistula
- Branchial fistula
- Thyroglossal fistula
- Tubercular sinus neck
- Median mental sinus.

Number: Openings may be single or multiple.

- Multiple sinus openings are seen in actinomycosis.
- Multiple fistula openings are seen in fistula in ano caused by tuberculosis, Crohn's disease.

Nature of discharge:

- Thin caseous (Tubercular)
- Thin watery on face (parotid fistula)
- Thick purulent (Bacterial infection)
- Yellow sulphur granules (Actinomycosis)
- Stools (Fecal fistula).

Surrounding skin:

- Bluish discoloration—tuberculosis
- Erythematous with cellulitis—acute infection
- Pigmentation—chronic sinus.

Palpation: Look for local tenderness, induration, direction of the tract, mobility of the tract on underlying structures and nature of discharge on pressure.

Adjoining structures should be palpated, e.g.

- Matted lymph nodes felt in tubercular sinus.
- Thickened underlying bone is felt in chronic osteomyelitis (Fig. 5.6).

Examination of draining lymph nodes:

- Firm and matted in tuberculosis.
- Firm, discrete and mildly tender in chronic nonspecific infection.
- Hard and fixed in malignancy.

General Examination

For malnutrition, diabetes, anemia, tuberculosis.

Specific Examination

- Oral cavity—in submental sinus
- Adjoining bones—in osteomyelitis
- Anal canal and rectum—in fistula in ano.



Fig. 5.6: Non-healing sinus forehead due to osteomyelitis of vault

Investigations

- Hemoglobin
- Urine
- TLC/DLC
- ESR—raised in chronic infections
- Blood sugar—for diabetes
- ELISA for HIV
- *Examination of discharge:*
 - Actinomycosis: Sulphur granules on gross-examination.
 - Bacterial infection: Gram staining, culture and sensitivity.
 - Tuberculosis: Z-N. staining for AFB, polymerase chain reaction (PCR) for tuberculosis.
- X-ray of the part: Osteomyelitis of underlying bone, radiopaque foreign body.
- Sinogram/fistulogram: To outline the tract to determine its course and relation with adjoining organs.
- Biopsy from margin of sinus: Confirms tuberculosis, malignancy.

ULCER

An ulcer is a break in the continuity of epithelial surface (skin or mucus membrane) due to microscopic tissue destruction. The dead tissue (slough) gets separated from the live tissue and exposes the floor of the ulcer.

Classification

1. *Nonspecific ulcer:* Their causes are given in Box 5.2.
2. *Specific ulcer:* Causes are tuberculosis, actinomycosis, syphilis.

3. *Malignant ulcer*: Causes are
- Squamous cell carcinoma
 - Basal cell carcinoma
 - Malignant melanoma

Life History of an Ulcer

It has following stages:

- Stage of extension*: The ulcer is progressive and growing in size. The ulcer has:
 - Sloughed floor
 - Indurated base
 - Purulent discharge
- Stage of transition*: The ulcer prepares for healing. The ulcer has:
 - Clear floor
 - Decreased induration of base
 - Serous discharge.
- Stage of repair*: The ulcer is nearly healed. The ulcer has:
 - Fibrous tissue on floor
 - No induration of base with healing margins
 - No discharge.

Box 5.2: Nonspecific ulcer—causes

Infective ulcer: Secondary bacterial infection of wounds.

Traumatic ulcer: Due to

- Mechanical trauma
 - Dental ulcer due to ill fitting dentures.
 - Decubitus ulcer due to pressure sores.
- Physical agents like burns, radiations.
- Chemical agents like acids and alkalis.

Trophic ulcer: Due to impaired tissue nutrition that depends upon blood supply and nerve supply.

- Arterial ulcer: Due to poor blood supply, e.g. Buerger's disease, Atherosclerosis.
- Venous ulcer: Due to venous stasis, e.g. varicose veins, deep vein thrombosis
- Neurogenic ulcer: Due to sensory impairment, e.g. diabetes, leprosy, tabes dorsalis. Also called as 'perforating ulcer'.

Tropical ulcer: Infective leg ulcers in tropical countries.

Diabetic ulcer

Cryopathic ulcer: Due to chilblains and cold injury

Mortorell's ulcer: Hypertensive ulcer

Bazin's ulcer (Erythrocyanoid ulcer): Calf ulcer in young girls due to fat necrosis, sometimes cause tuberculosis.

Clinical Examination of an Ulcer

History

- *Duration of ulcer*: Short in acute ulcer and long in chronic ulcer.
- *Mode of onset*
 - ☐ Following trauma: Traumatic ulcer.
 - ☐ Following sexual contact: Syphilitic ulcer, chancroid.
 - ☐ Long standing varicose veins: Varicose ulcer.
 - ☐ Over a scar: Marjolin's ulcer.
 - ☐ Over matted lymph nodes in neck: Tubercular ulcer.
 - ☐ Over a nodule: Malignant ulcer.
- *Progress*: Change in size of ulcer.
- *Painful or painless*: Inflammatory and tubercular ulcers are painful, malignant and syphilitic ulcers are painless.
- *Nature of discharge*: Pus, blood, serum.
- *Constitutional symptoms*: Fever, cough, anorexia, weight loss.

Local Examination

- **Site**
 - ☐ Tubercular ulcer—in neck.
 - ☐ Rodent ulcer—upper part of face.
 - ☐ Arterial ulcer—tip of toes, dorsum of foot.
 - ☐ Venous ulcer—above medial malleolus.
 - ☐ Neuropathic ulcer—pressure points on sole.
- **Size**: Exact dimensions.
- **Shape**: Round, oval, irregular or serpiginous (healing at one place and extending at another place).
- **Edge** (Fig. 5.7)
 - ☐ Sloping—healing non-specific ulcer, venous ulcer.
 - ☐ Undermined—tubercular ulcer (bluish margins).
 - ☐ Raised and everted—squamous cell carcinoma.
 - ☐ Rolled out—rodent ulcer.
 - ☐ Punched out—syphilis.
- **Floor**: This is the exposed surface of the ulcer that can be seen. It can have:
 - ☐ Sloughed necrotic tissue—ulcer in stage of extension.
 - ☐ Red granulation tissue (Fig. 5.8)—healing ulcer in stage of transition.

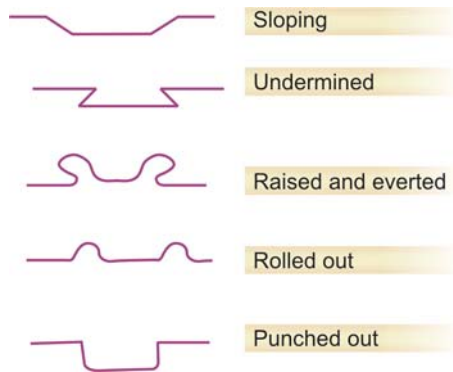


Fig. 5.7: Ulcer—shape of the edge



Fig. 5.8: Red granulation tissue on floor of the ulcer

- ☐ Pale smooth granulation tissue—ulcer in stage of healing.
- ☐ Wash leather slough—syphilitic ulcer.
- ☐ Watery or 'apple jelly' granulation tissue—tubercular ulcer.
- ☐ Floor raised above the surface—malignant ulcer.
- **Base:** It is the area on which ulcer rests. Move the edges of the ulcer between thumb and index finger so as to feel the *underlying tissues* (e.g. fascia, tendons, muscles, bone).
Feel for the *induration* of the base.
 - ☐ Mild induration felt in chronic nonspecific ulcer.
 - ☐ Marked induration felt in malignant ulcer, syphilitic ulcer.
 - ☐ Feel for the *mobility* of ulcer on underlying structures. Benign ulcers are usually mobile while malignant ulcers are fixed to underlying structures.

If on palpation, ulcer is friable and bleeds, it is likely to be malignant. However, healthy granulation tissue also bleeds on palpation.

- **Nature of discharge:** It can be scanty or copious.
 - ☐ Purulent discharge—bacterial infection.
 - ☐ Watery discharge—tuberculosis.
 - ☐ Bloody discharge—malignancy.
 - ☐ Sulphur granules—actinomycosis.
- **Surrounding area:**
 - ☐ Inflamed and edematous—infective ulcer.
 - ☐ Thick, pigmented with dilated veins—varicose ulcer.
 - ☐ Palpable matted lymph nodes—tubercular ulcer.
 - ☐ Pigmented halo—malignant melanoma.

Regional Examination

- **Draining lymph nodes**
 - ☐ Tender and enlarged—secondary infection.
 - ☐ Enlarged, hard, fixed—malignant ulcer.
 - ☐ Enlarged, firm, matted—tubercular ulcer.
 - ☐ Enlarged, shotty—syphilitic ulcer.
- **Examination for impaired circulation:** Look for weak or absent arterial pulsations with trophic changes (thin limb, shiny skin, loss of hair, brittle nails).
 - ☐ Look for varicose veins
- **Examination for neurological deficit**
Look for: Sensations
Motor power
Reflexes

General Examination

For anemia, malnutrition, jaundice, diabetes.

Systemic Examination

- Respiratory system—Pulmonary tuberculosis.
- CNS and spine—Neuropathic ulcer.
- CVS—Congestive heart failure, valvular defects.

Investigations

- **Hemoglobin**—to look for anemia.
- **TLC/DLC**—count raised in secondary infection.
- **ESR**—raised in chronic infection.
- **PBF**—to look for sickle cell anemia.
- **Blood sugar**—for diabetes.
- **Serology for syphilis**—VDRL, Kahn test.
- **X-ray chest**—For pulmonary tuberculosis.

- **Examination of discharge:**
 - Gram staining for bacterial infection.
 - ZN stain for AFB, culture and sensitivity.
 - PCR of discharge for tubercular infection.
- **X-ray of affected part**—osteomyelitis of underlying bone.
- **FNAC of enlarged draining lymph nodes** can show tuberculosis, malignancy.
- **Ulcer biopsy :**
 - *Wedge biopsy* from margin of ulcer including adjoining normal tissue as well. The biopsy is not taken from the center of the ulcer as it contains mainly necrotic material.
 - *Excision biopsy* is done in case of small ulcer and subjected to histopathological examination.

Treatment

Treatment during stage of extension:

- **Antibiotics** according to culture and sensitivity report of the pus discharge.
- **Analgesics** and **anti-inflammatory drugs** for control of pain and inflammation.
- **Bed rest** and **limb elevation** to relieve pain and edema in leg ulcers.
- **General measures** like:
 - Correction of anemia by hematinics/ blood transfusion.
 - High protein diet with vitamins (vitamin C) to improve nutrition and wound healing.
 - Control of diabetes (if present).
- **Local (topical treatment):** It is done with regular antiseptic dressings. The aim is to remove slough and control sepsis so that healthy granulation forms and epithelialization starts. *Various methods* are:
 - *Eusol (Edinburgh University solution)* is used for desloughing of wound. It contains boric acid and bleaching power.
 - Solutions releasing nascent oxygen make bubbles in the wound and help in separation of slough, e.g. H₂O₂, Oxum solution.
 - *Magnesium sulphate (Sumag) ointment* is hygroscopic in nature and applied on area surrounding the ulcer. It helps in relieving local edema and cellulitis.
 - Once line of demarcation appears between slough and healthy tissue, *mechanical debridement* should be done in multiple sittings.
- *Regular wound dressings* are done using antiseptic solution or local antibiotics. However, these should be used only till the infection becomes clear. Their excess use can interfere with normal healing because they are toxic to fibroblasts and resistant strains of bacteria may develop.
- *Steps of dressing* are:
 - a. Wound cleaning with sterile and warm saline solution.
 - b. Application of local antiseptic, e.g. Povidone iodine, chlorhexidine, mupirocin etc.
 - c. Covering the ulcer with sterile gauge pieces.
 - d. Putting cotton pads to absorb the discharge.
 - e. Applying bandage.

The dressing is changed once or twice a day depending upon soakage due to discharge.

The features of an ideal dressing are given in Box 5.3.

Other agents used for dressing of ulcers are:

- i. **Hydrocolloids:** It is made of polyurethane foam that expands and forms a gel in the wound. It promotes angiogenesis and wound healing.
- ii. **Alginates:** These are sodium and calcium salts of alginic acid. These are hemostatic and useful in management of bleeding wounds. They absorb liquids and swell to form gel, hence, useful in heavy exuding wounds.
- iii. **Tegaderm:** It is thin polyurethane membrane that prevents water loss from the ulcer. It prevents contamination of ulcer from the environment. Epithelial regeneration occurs rapidly and it prevents epithelial breakdown due to friction or exposure. It is useful in treating non-oozing wounds.
- iv. **Recombinant epidermal growth factor:** It increases collagen production and stimulates formation of granulation tissue. Thus, it enhances wound healing and reduces healing time. It is useful in dressing of clean wounds.

Box 5.3: Ideal dressing

- Removes exudates and toxins
- Maintains high humidity in the wound
- Porous (permits gaseous exchange with atmosphere)
- Non-allergic
- Non-irritant
- Non-toxic
- Easy to remove (without causing trauma)
- Cost effective

Treatment during Stage of Transition

- At this stage, ulcer is having healthy granulation tissue and minimal discharge.
- Aim is to promote surface epithelialization and to prevent secondary wound infection.
- Non-adhesive sterile dressing is done on alternate days or even twice a week using vaseline gauze. It helps in easy removal of dressing and prevents epithelial breakdown during change of dressing.
- If there is formation of hypergranulation tissue (proud flesh), it is debrided surgically or by application of copper sulphate (chemical cautery).
- Small ulcers heal of their own while large ulcers need coverage with skin grafting or flaps.

Treatment of Underlying Cause

- Varicose ulcer—surgery for varicose veins.
- Tubercular ulcer—antitubercular treatment.
- Malignant ulcer—wide excision.
- Diabetic ulcer—control of diabetes.

CLINICAL FEATURES OF VARIOUS ULCERS**Traumatic Ulcer (Fig. 5.9)**

- Can occur anywhere in the body.
- More common in areas prone to trauma (bony prominences like shin, malleoli, heel, tongue).
- Small, painful and circular ulcers.
- Repeated trauma on shin prevents ulcer healing (Footballers ulcer).

**Fig. 5.9:** Traumatic ulcer tongue due to sharp tooth**Fig. 5.10:** Arterial ulcer**Arterial Ulcer (Fig. 5.10)**

- It is due to inadequate skin perfusion due to peripheral arterial disease.
- Common causes are atherosclerosis, Buerger's disease, diabetes.
- Commonly seen in parts prone to trauma (anterior and lateral side of leg, toes, heel, dorsum and sole of foot).
- Hallmark of arterial ulcer is rest pain.
- Ulcers are irregular, punched out and deep (involving deep fascia, tendon or even bone).
- The affected limb shows gangrenous patches and trophic changes (See Chapter 18: Gangrene and Diseases of Arterial System).
- The limb feels cold and peripheral pulses are diminished or absent.
- Investigations helpful in diagnosis are: Doppler pressures, duplex ultrasonography and arteriography.

Venous Ulcer (Fig. 5.11)

- It is typically situated on medial side of lower half of the leg above medial malleolus.
- It is vertically oval in shape, sloping edges and never penetrates deep fascia.
- It is painless.
- Surrounding skin shows varicose veins, pigmentation and eczema (Lipodermatosclerosis). (Details of venous ulcer are given in chapter 19: Diseases of Venous System).



Fig. 5.11: Venous ulcer



Fig. 5.12: Neurogenic ulcer sole

Box 5.4 : Comparison between arterial and venous ulcer

	<i>Venous ulcer</i>	<i>Arterial ulcer</i>
Symptoms	Painless	Rest pain
Previous history	Varicose veins, DVT	IHD, Smoking, diabetes
Site	Medial or lateral side of leg above malleoli	Foot, shin
Number	Single	Multiple
Shape	Oval	Irregular
Edges	Sloping	Punched out
Depth	Shallow (does not penetrate deep fascia)	Deep (penetrates deep fascia)
Discharge	Sloughing with high exudates	Low exudates
Surrounding area	Varicose veins, Lipodermatosclerosis	Trophic changes cold limb
Pulses	Normal	Diminished or absent
Ankle-brachial pressure index (ABPI)	> 0.8	< 0.6

- Comparison between arterial and venous ulcer is given in Box 5.4.

Neurogenic Ulcer

- It is due to repeated trauma or pressure in an area that has lost sensations.
- Causes are diabetic neuropathy, paraplegia, leprosy, peripheral nerve injury.



Fig. 5.13: Neurogenic ulcer lateral malleolus

- Common sites are:
 - Heel and ball of the foot (in ambulatory patients) (Figs 5.12 and 5.13) .
 - Buttocks and back of the heel (in bedridden patients).
- Bed sores and trophic ulcers are typical examples (See Chapter 18: Gangrene and Diseases of Arterial System).
- Ulcers are painless because of anesthesia.
- It starts as a callosity that gets infected and discharges pus leading to ulcer formation.
- The ulcer gradually burrows through deeper tissues and reaches up to bone causing osteomyelitis. Hence, it is also called 'Perforating ulcer'.
- Neurological examination reveals loss of sensations and diminished motor power.
- Peripheral pulses are well palpable.

Tropical Ulcer (Phagedenic Ulcer)

- It is seen in tropical countries.
- It is caused by Vincent's organisms (*Borrelia vincentii*).
- Following minor trauma, pustule develops that bursts and spreads rapidly forming big ulcer.
- There is constant pain and odorous discharge from the ulcer.
- There are minimal constitutional symptoms.
- The edges of the ulcer are undermined, slough is present on floor and there is copious discharge.
- Often there is acute lymphadenitis.
- The ulcer refuses to heal for months and years.
- Healing occurs with formation of pigmented scar.

Diabetic Ulcer (Fig. 5.14)

- It is commonly seen in toes and feet.
 - There is associated sepsis of adjoining tissue.
 - Neglected cases may develop gangrene of toes and feet.
 - Etiological factors include:
 - Angiopathy leading to local ischemia.
 - Neuropathy leading to impaired sensations.
 - Decreased resistance to infection.
- (Details are given in Chapter 18: Gangrene and Diseases of Arterial System).

Tubercular Ulcer (Fig. 5.15)

- Commonly seen in neck following bursting of cold abscess.
- It may develop from tuberculosis of bones and joints.



Fig. 5.14: Diabetic ulcer involving foot and leg



Fig. 5.15: Tubercular ulcer with underlying cold abscess neck

- Ulcer is usually painful.
- Ulcer is oval in shape with irregular borders.
- Margins are undermined with bluish discoloration.
- The ulcer is shallow with pale granulation tissue on floor and serosanguinous discharge.
- The base is indurated and fixed to underlying structures, e.g. matted lymph nodes, bone, joint, etc.
- Multiple scars are usually seen in adjoining skin due to healed lesions.
- Cutaneous tuberculosis of face is called 'Lupus Vulgaris' (See Chapter 4: Specific Infections).

Syphilitic Ulcer (Gummatous Ulcer)

- Ulcers in syphilis are seen in all the three stages—primary, secondary and tertiary syphilis. (Details are given in Chapter 4: Specific Infections).

Actinomycosis

- It leads to formation of multiple ulcers on an indurated base.
- The surrounding skin shows bluish discoloration.
- The discharge typically contains sulphur granules. (Details are given in Chapter 4: Specific Infections).

Squamous Cell Carcinoma (Fig. 5.16)

- It may occur anywhere but common sites are lips, cheeks, tongue, anus, penis.
- Ulcer is irregular in shape.
- Edges are raised and everted.
- Floor is covered with irregular necrotic tumor and granulation tissue.



Fig. 5.16: Malignant ulcer with everted margins—
Squamous cell carcinoma



Fig. 5.17: Basal cell carcinoma at outer canthus of eye

- Base is indurated. In early stage, it is mobile on underlying structures. In advanced cases, it invades and gets fixed to underlying structures.
- Regional lymph nodes are enlarged due to metastasis or due to secondary infection.
(Details are given in Chapter 11: Tumors).

Rodent Ulcer (Basal Cell Carcinoma) (Fig. 5.17)

- Mostly seen on sun exposed area of face (above line joining angle of mouth with ear lobule).
- Ulcer is rounded in shape.
- Edges are raised and rolled out.
- The floor is covered with a coat of dried serum and epithelial cells that bleeds on scratching.
- Base is indurated and fixed to deep structures like muscles, bone, etc.
- Minute venules in the edge are characteristic.
- Regional lymph nodes are not enlarged.
(Details are given in Chapter 11: Tumors).

Marjolin's Ulcer (Fig. 5.18)

- It is squamous cell carcinoma arising in scar or chronic benign ulcer.
- Scar undergoing malignant change is usually post burn scar.



Fig. 5.18: Marjolin's ulcer developing in
chronic venous ulcer

- Chronic ulcer becoming malignant is usually venous ulcer.
- It is painless due to lack of nerve supply and often ignored by the patient.
- It is slow growing due to less vascularity.
- Edges of ulcer are not always raised and everted.
- There is no lymphatic metastasis as lymphatics are destroyed.
(Also see Chapter 11: Tumors).

6

Chapter

Wounds

Sanjay Marwah

DEFINITION

Break in continuity of lining surface epithelium is defined as wound. Wound is seen in a wide variety of situations, e.g. after an accident, assault, surgery and even self inflicted wound.

From practical point of view, wounds are classified into **tidy** and **untidy wounds**.

Tidy Wounds

These are clean wounds caused by sharp instruments and can be closed primarily. If underlying structures (nerves, vessels, etc.) are damaged, they can be repaired at the same sitting before wound closure.

Untidy Wounds

These are soiled wounds caused by crushing and avulsion injuries. The underlying structures (nerves, vessels, etc.) are crushed to variable extent. They cannot be closed primarily because in presence of foreign bodies and devitalized tissues, there are high chances of wound infection, wound dehiscence, septicemia and even death. The treatment is wound toilet and excision of all dead tissues so that it gets converted to a tidy

wound. Then it can be closed primarily or allowed to heal with secondary intention.

The classification of surgical wounds is given in Table 6.1.

TYPES OF WOUND (TABLE 6.2)

Abrasion, Contusion and Hematoma

In **abrasion**, there is irregular tearing of only superficial layers of skin as body skids on a rough surface (like road). Bleeding points and sensitive nerve endings are exposed leading to severe pain. At the same time, dirt gets embedded in the wound (Fig. 6.1). Treatment largely consists of prevention of infection by scrubbing the wound with soap and water and sterile dressing. Abrasions of face may be left uncovered. Healing occurs in about 10 day's time. In case of infection full thickness skin loss may occur.

In **contusion**, skin surface remains intact and subcutaneous bleeding occurs leading to swelling and skin discoloration (Fig. 6.2). It usually occurs following a blow. The color is initially red, turning gradually to blue and then black. Finally, it fades to greenish yellow and to normal skin color. No treatment is required for contusion.

Table 6.1: Classification of surgical wounds

Class	Definition	Infection rate
Clean	Wound of elective surgery where hollow viscera* are not entered, e.g. lymph node biopsy.	2%
Clean contaminated	Wound where hollow viscera* are entered with minimal contamination.	10%
Contaminated	Wound where hollow viscera* are entered with uncontrolled spillage.	20%
Dirty	Wound with pus in operative field, e.g. abscess drainage.	> 50%

*Hollow viscera—gut, respiratory tract, genitourinary tract

Table 6.2: Types of wounds and their management

Types of wound	Description	Management
Abrasion	Injury of superficial skin layers.	Wound toilet, dressing
Contusion	Small subcutaneous hematoma.	Conservative
Hematoma	Large blood collection.	Needs drainage
Incised wound	Clean wounds.	Primary closure
Lacerated wound	Irregular, contaminated, deeper tissues crushed.	Toilet and debridement, delayed closure.
Punctured wound	Deep contaminated wounds with small skin opening. Vital structure may be injured.	Needs observation/intervention.
Avulsion wound	Degloving injury raising skin flaps.	Wound toilet and loose stitching.
Crushed wound	Edema and bleed in closed fascial compartments, tissue ischemia, limb loss, renal failure (Compartment syndrome).	Early fasciotomy is required.

In **hematoma**, there is more severe injury leading to collection of large volume of blood in tissue planes (Fig. 6.3). Small hematomas are usually reabsorbed, but large hematomas need intervention otherwise complications may occur.

- A hematoma may get infected leading to abscess formation that requires incision and drainage.
- A hematoma may liquefy producing a cystic swelling (seroma) that can be aspirated with a wide bore needle. Sometimes repeated aspirations are required till it resolves completely.



Fig. 6.1: Abrasion thigh



Fig. 6.2: Contusion forearm



Fig. 6.3: Hematoma cheek

- A large hematoma making a clot and producing pressure effects (e.g. intracranial hematoma) should be promptly evacuated by surgical intervention.
- A hematoma in a muscle may organize into fibrous tissue producing a very firm swelling. It may be replaced by calcifying osteoid tissue (**myositis ossificans**) typically seen in quadriceps femoris muscle.

Incised Wounds

These are mainly caused by sharp knife, metal and glass. These are relatively clean wounds and injury occurs along the track of penetration only. After thorough cleaning, wound should be explored to look for any injury to deeper structures. Damaged nerves, vessels and tendons should be repaired. The incised wound is ideal for primary closure if done within 6 hours of injury.

Lacerated Wounds

These are irregular and untidy wounds caused by crushing and tearing forces (Fig. 6.4). There is contusion and abrasion of surrounding area. In the depth of the wounds, the nerves and vessels may be stretched and torn rather than cleanly divided. Mostly these wounds are grossly contaminated with dust and foreign materials. There is rapid proliferation of bacteria in dead and devitalized tissues leading to infection. Treatment is thorough wound toilet, excision of dead tissue and primary closure if done within 6 hours of injury. If treatment is delayed, the wound should be left open and repaired after a few days when edema and inflammation has subsided.



Fig. 6.4: Laceration cheek

Punctured Wounds

These wounds are deeper than their length. These are caused by stabbing action of a long, thin weapon (like sword) or by a missile (like bullet). There is risk of injury to deeper organs as well as infection due to contamination along the track of the wound.

A punctured wound can be:

- Penetrating wound*: It is an entry wound only.
- Perforating wound*: It has both entry and exit wound.

All punctured wounds in neck, chest and abdomen are potentially lethal. Cases with such wounds should be hospitalized and thoroughly investigated. In case, injury to vital structures is suspected, early exploration should be done.

In missile injuries, degree of damage depends upon the velocity of bullet with a low velocity bullet, injury occurs in a straight tract and surrounding structures are not damaged. High velocity bullets create shock waves while passing through the tissues. It causes widespread tissue destruction due to cavitation effect (Fig. 6.5). After stabilization of vital signs, the bullet wound is treated by exploration (Box 6.1).

Box 6.1 : Management of bullet wounds in limbs

- Liberal skin incision.
- Thorough wound toilet.
- Identification of neurovascular bundle.
- Excision of all dead tissues.
- Hemostasis.
- Leave wound open.

Avulsion Wounds

These are caused by shearing force that detaches the skin from its underlying structures. The raised skin flap may remain attached at one edge; the so called

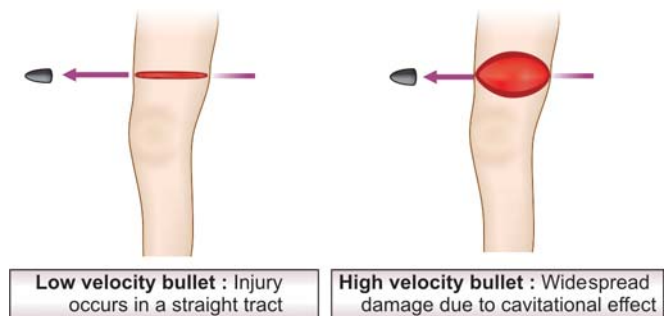
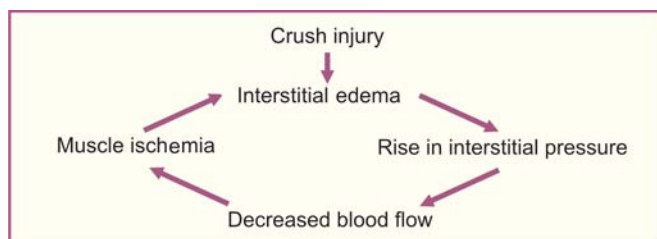


Fig. 6.5: Difference in damage produced by low and high velocity bullet

Box 6.2: Pathophysiology of crush injury



degloving injury. The most common plane of separation is between subcutaneous fat and deep fascia.

Such injuries are commonly seen in factory workers when long hair or skin is caught in the roller machines. The treatment is thorough cleansing of the wound, excision of obvious dead tissues and reposition of skin flap with a few stitches. The risk of avulsion injury is that devascularized skin flap will slowly necrose and become dead. If skin flap is completely detached and crushed, then wound requires skin grafting after thorough toilet and debridement of dead tissues.

Crushed Wounds

There is a severe blunt injury to the tissues leading to inflammation, edema and bleeding within closed fascial compartments. It is usually seen in war injuries, earthquakes and severe road side accidents. There is rise in interstitial pressure leading to decreased blood flow and muscle ischemia that further adds to interstitial edema (Box 6.2). Thus, a vicious cycle starts leading to progressive ischemia of muscles and nerves and then limb loss (**Compartment syndrome**). Ischemic muscles release myoglobin into circulation that can cause acute renal failure.

In compartment syndrome, peripheral pulses are usually palpable. The most important clinical sign is that passive stretching of the affected limb leads to worsening of pain. The treatment is urgent fasciotomy. Longitudinal incisions are given on skin and deep fascia so that compressed muscles are released and their circulation is restored. If a case of crush injury reports after several days of trauma, the muscles are already dead and there is no chance of recovery. The fasciotomy in such a situation will cause myoglobinuria and renal shut down. Hence, amputation of dead limb rather than fasciotomy is a safer option in such case.

WOUND HEALING

- If wound edges are approximated as is done in a clean incised wound, rapid healing occurs and a thin

scar is produced. It is called as **healing by primary intention**.

- If wound edges cannot be approximated due to presence of devitalized tissue, infection or skin loss, healing process becomes slow. It produces large unsightly scar with restricted movements due to contractures. The lining epithelium of scar is weak and undergoes repeated ulceration following trauma. It is called as **healing by secondary intention**.

Phases of Wound Healing

1. *Phase of inflammation (Day 1-4):* It is also known as "Lag phase". Injury results in bleeding and the blood comes in contact with collagen tissue and activates kinins and complement cascade. Clotting factors are activated and platelets aggregate leading to blood clot formation. Capillary permeability increases leading to escape of RBC and WBC into the wound. Polymorphs act as scavengers by removing dead tissue during initial 48 hrs. Then monocytes come into action and act as scavengers from 3rd to 5th day. By 5th day, capillary budding and fibroblast proliferation starts leading to next phase of granulation tissue formation.
2. *Phase of granulation tissue (Day 5-20):* The granulation tissue is rich in fibroblasts that secrete collagen and ground substance. The fibroblasts produce procollagen (immature form) that gets converted to collagen (mature form) by hydroxylation. The mature collagen fiber gives strength to the tissues. Ground substance is a thin gel like binding agent that binds the collagen fibers.
3. *Phase of scar formation (Day 20 onwards):* In this phase remodeling of haphazardly arranged collagen fibers takes place. New collagen fibers are synthesized in an orderly fashion along lines of tension in the scar. Vascularity becomes less and ingrowth of nerve fibers and lymphatics takes place. There is imperceptible scar remodeling and gain in strength continues up to 2 years. Hence, scar revision for cosmetic reasons should not be done before 1 year.

Repair of Surface Epithelium

Epithelium starts growing and migrating towards the wound from skin edges in 12 hrs. The wound epithelialization is usually complete in 48 hrs in incised wound. However, there is no regeneration of sweat, sebaceous glands and hair follicles in the new epithelium.

Adverse factors for wound healing are given in Box 6.3.

Box 6.3: Adverse factors for wound healing

General factors	Local factors
Old age	Wound hematoma
Anemia	Wound infection
Hypoproteinemia	Necrotic tissue in wound
Uremia	Foreign material in wound
Diabetes	Poor blood supply
Jaundice	Tension on suture line
Malignancy	Faulty wound closure
Chemotherapy	Lack of rest to the sutured area
Steroids	Local radiotherapy
Immunodeficiency (HIV infection)	

EXAMINATION OF WOUNDS

- First examine the patient as a whole and look for vital signs—pulse, blood pressure, respiration, consciousness level, temperature, etc.
- Examine the wound (See Table 6.2).
- Examine structures deep to the wound viz:

In limbs

- ☐ Look for major vessel injury by feeling peripheral pulses.
- ☐ Look for tendon injury by testing movements.
- ☐ Look for nerve injury by testing sensations and movements.
- ☐ Look for any fractured bones.

In head Look for injuries to skull, brain, eyes and ears.

In chest Look for injuries to lungs, heart and great vessels.

In abdomen Look for injury to solid and hollow viscus.

TREATMENT OF WOUNDS

- General management of the injured patient for maintenance of airway, breathing and circulation (See chapter 10—Care of the acutely injured).
- Anesthesia is required for complete examination and surgical toilet of the wound. Most minor wounds can be treated under local anesthesia with a regional block. 2% lignocaine is infiltrated into the tissues around and beneath the wound with a 23 G needle.

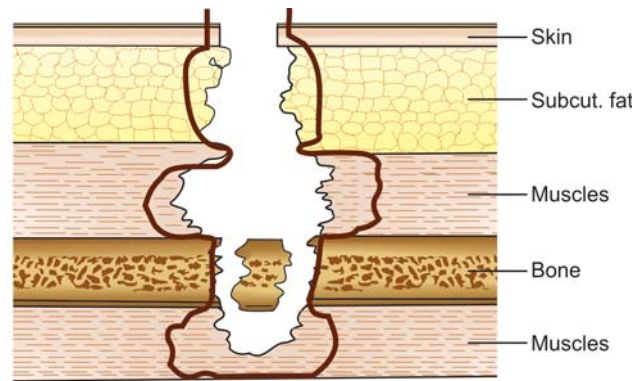


Fig. 6.6: Wound excision

- In major wounds especially in children, general anesthesia is needed.
- In case of a limb wound having severe bleeding, a tourniquet may be applied in upper arm and upper thigh to obtain bloodless operative field. The pressure in tourniquet is raised above systolic blood pressure for not more than 45 minutes.
- The surrounding skin as well as wound is cleaned with a detergent solution to remove dead and foreign material.
- The wound is explored to assess the extent of damage. Sometimes the wound margins need extension on both sides to help complete the exploration (in punctured wounds).
- A clean, superficial, incised wound undergoes primary repair with sutures.
- A heavily contaminated, lacerated and deep wound needs debridement of devitalized tissues called as **wound excision** (Fig. 6.6).
- Excision starts with superficial layers and then extends to deeper structures. Only minimal amount of skin should be removed especially in hands and face.
- Deeper structures like nerves, vessels and tendons in the wound are exposed and left in continuity.
- Dead fat (pink), dead muscle (dark colored) and loose bone fragments are excised. Tissue excision is continued till normal bleeding is observed and the wound starts looking like an anatomical dissection.
- If there is no significant loss of tissues and margins can be approximated without tension, **primary repair** can be done **after wound excision**. Deeper layers are approximated with absorbable sutures. Aim is to obliterate the dead spaces without causing

undue tension on the tissues. Skin is approximated with sutures, clips or staples.

- In case of edematous tissues with questionable viability primary wound closure will lead to tissue ischemia and sepsis (gas gangrene). Such wound should be left open and dressed. The edema usually subsides in 4-6 days and tissues can be approximated without tension. Closure at this stage is called **delayed primary repair**.
- In case of gross sepsis of the wound, it is left open and treated with regular dressing and antibiotics. It takes around 2 weeks time in becoming clean. Closure of the wound at this stage is called as **secondary suturing**.
- In case of wound with skin loss, plastic reconstruction is done with skin graft, skin flap or free tissue transfer after wound becomes clean.

MANAGEMENT OF FACIAL WOUNDS

- Facial wounds bleed profusely due to high vascularity.
- Facial artery can be tied safely without any risk of ischemic tissue damage due to rich collateral circulation.
- Careful clinical examination should be done to rule out injury to deeper structures like facial nerve and parotid duct.
- Inside of the mouth should always be examined.
- In case of suspected fracture or retained foreign body, X-ray is indicated.
- Ragged skin edges need minimum trimming.
- Deep tissues are repaired with absorbable sutures.
- Skin is accurately approximated with several, fine, non-absorbable sutures.
- Wounds crossing linear features must be accurately approximated to avoid deformity, e.g. in lip injury, red margin should be sutured accurately.
- A wound inside mouth should be sutured first before suture of external wound to avoid tearing of external sutures.
- Skin sutures are removed on 4th day since wound heals rapidly due to good blood supply and less cross marks are produced by stitches due to early removal.

MEDICOLEGAL ASPECTS OF WOUNDS

The injuries caused by wounds are classified as **simple**, **grievous** or **dangerous to life**.

Simple Injury

It is defined as an injury:

- Which is neither extensive nor serious to the sufferer.
- Which bleeds minimally.
- Which heals rapidly without leaving any permanent disability or disfigurement to the sufferer, e.g. abrasions, contusions.

If simple injury occurs following assault, it is covered under section 323 of Indian Penal Code (IPC), i.e. punishment for voluntarily causing hurt. The accused can be awarded imprisonment for one month to one year.

Grievous Injury

Broadly speaking, grievous injury is one that causes permanent disability or disfigurement to the sufferer. The following kinds of hurt are designated as grievous:

- i. Emasculation, i.e. depriving man of his virility (damaging testicles).
- ii. Permanent loss of vision in one or both eyes.
- iii. Permanent deafness of either ear.
- iv. Permanent loss of any organ, limb or joint.
- v. Permanent impairment of power of any limb or joint.
- vi. Permanent disfiguration of the head or face.
- vii. Fracture or dislocation of a bone or tooth.
- viii. Any hurt which endangers life or causes the sufferer a severe bodily pain for 20 days when he is not able to follow ordinary pursuits (sleeping, walking, eating etc.).

The grievous injury following assault is covered under section 320 IPC (grievous hurt) and can award upto seven years of imprisonment to the accused.

Dangerous to Life

A dangerous injury is a type of grievous injury and is defined as one which poses immediate danger to life of the sufferer by direct effect of injury, e.g. compound fracture skull, penetrating injury over any of three great body cavities (cranium, pleura and peritoneum), rupture of internal viscera (liver, spleen, etc.).

The dangerous injury occurring after assault is covered under section 307 IPC (attempt to murder) and can award up to ten years of imprisonment. In case of death following injury, it is covered under section 302 IPC (punishment for murder) and can award life imprisonment or even death sentence.

The **injuries occurring after accidents** (rash driving, negligent conduct) are covered under section 284 IPC (accidental injuries). In case of death occurring after accident or professional negligence (e.g. death following negligence during surgery), it is covered under section 304 A and can award imprisonment up to two years.

COMPLICATIONS OF WOUND HEALING

1. *Wound infection*: The patient complains of throbbing pain and on examination, the wound is tense and inflamed. The skin sutures need to be removed and wound laid open to allow free drainage of pus. Pus culture sensitivity is done. The wound is dressed regularly and appropriate antibiotics are given. Once all the inflammation disappears, secondary suturing can be done.
2. *Hypertrophic scar*: All wounds heal by scarring. The stages in formation of scar are:
 - i. Healing (0-4 weeks): The scar is fine, soft, not contracted and not strong.
 - ii. Remodeling (4-12 weeks): The scar is red, raised, itchy, tender and starts contracting.
 - iii. Maturation (12-40 weeks): The scar becomes soft, supple and white and tends to relax.

Box 6.4: Factors for ideal scar

- Clean incised wound.
- No tension on suture line.
- Healing with primary intention.
- Incision along skin crease.
- Old person.
- Lax skin.
- Site: Vermillion border, eyelid.

Factors helpful in producing ideal scar are given in Box 6.4. If the scar remains in remodeling stage for a longer time, it is called as **hypertrophic scar** (Fig. 6.7). It remains red, raised, itchy and tender usually up to six months and then gradually regresses. Application of moisturizing cream and pressure garments can accelerate the process of scar maturation.

3. *Keloid (like a Claw)*: There is excessive growth of the scar tissue so that it spreads like a claw into adjoining normal tissues that are not affected by original wound (Fig. 6.8). It has itching,



Fig. 6.7: Hypertrophic scar shoulder



Fig. 6.8: Keloid sternum

erythematous and spreading margins. The keloid continues to grow even after 1 year of injury and sometimes even progress for 5-10 years. It appears that maturation and stabilization of collagen fibers is inhibited. The common site for keloid is sternum, back and shoulders; and is seen more commonly in Negroes.

Treatment is extremely difficult. Surgical excision is usually followed by recurrence. Use of pressure garments and intralesional injection of triamceno-lone with hyalase might help in controlling its growth.

Differences between hypertrophic scar and keloid are given in Box 6.5.

4. *Skin pigmentation*
5. *Contractures*: Since wound contraction continues during scar maturation so final scar is always shorter than original wound. The scar should be placed parallel to the line of wrinkle so that on healing, it

Box 6.5: Hypertrophic scar vs keloid

<i>Hypertrophic scar</i>	<i>Keloid</i>
Nonfamilial	Familial
No relation with race	More in black race
Young children	Females
Subsides after 6 months	Continues to grow even after 1 year
On flexor surfaces	On sternum, shoulder, back
Doesn't spread to normal tissues	Spreads to adjoining normal tissues
No active treatment needed	Difficult to treat, surgery leads to recurrence
Treatment: Moisturizing cream, pressure garments.	Treatment: Pressure garments, local steroid injections

looks like another wrinkle. On face and neck, the lines of wrinkles are at right angles to the direction of fibers of underlying muscles (Fig. 6.9). So linear scars cutting the lines of wrinkles will lead to

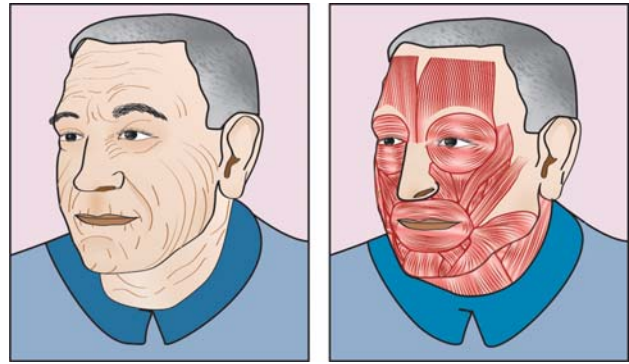


Fig. 6.9: Line of wrinkles at right angle to line of muscles

contracture formation and restricted mobility. It is more common if healing occurs with secondary intention, e.g. post-burn contractures. The treatment is by doing plastic procedures like Z plasty, Y-V plasty or scar excision with skin grafting.

6. *Marjolin's ulcer*: Squamous cell carcinoma developing in long standing scar is called as Marjolin's ulcer (Chapter 5: Sinus, Ulcer and Fistula and Chapter 11: Tumors).

7

Chapter

Surgical Asepsis and Antiseptic Measures

Sanjay Marwah

Surgical asepsis is defined as prevention of access of microorganisms to a surgical wound.

Antiseptic measures are the measures used to destroy bacteria or remove them from all objects coming in contact with the wounds.

Surgical wound sepsis can be prevented/minimized by:

- Following operation theater (OT) discipline
- Proper instrument sterilization

OPERATION THEATER DISCIPLINE

- OT discipline should begin in the ward itself and should be followed till the patient is shifted back to the ward.
- The operative area is shaved and patient should take bath with soap and water on the evening before surgery and should wear dry and clean OT dress.
- The entry points of OT should be separate for the patients and doctors.
- All persons entering theater complex should wear clean overshoes.
- Change your clothes with theater clothing made of cotton and freshly laundered.
- Disposable caps and masks are used to minimize risk of contamination from hair, nose and mouth.
- The patient is brought to theater on a ward trolley and shifted to theater trolley in reception area and then taken to the operation table.
- The movement of theater personnel in and around the operating room should be kept to a minimum.
- The operating room should have unidirectional (laminar) air flow system that helps in decreasing the number of bacteria to which patient is exposed.
- Scrubbing of the hands and forearms (up to elbows) by the surgical team (surgeon, nurse and assistant).



Fig. 7.1: Elbow tap to control water flow

Special sinks are designed for this purpose to allow adequate flow of warm water and water flow is controlled with elbow taps (Fig. 7.1). Antiseptic solutions (povidone iodine, chlorhexidine with ethyl alcohol) are used for scrubbing and soft brushes are used for cleaning the nails. Wash is done for 8-10 minutes by watch for first surgery and for 5 minutes for subsequent surgeries. At the end of scrub, hands and forearms are rinsed with running water and then hands are held up above the elbow level so that any remaining water on hands flows down with gravity. The tap is turned off and hands and elbows are dried with a towel.

- Surgical gown is put on and it acts as a barrier from surgeon to the patient and vice-versa. Disposable gown is better for an implant surgery for minimizing



Fig. 7.2: Unrolling of sterile gown from upper end and inner surface



Fig. 7.4: Wearing right glove—Glove held from inside with left hand



Fig. 7.3: Method of wearing gown



Fig. 7.5: Wearing left glove—Glove held from outside with right hand

infection. The upper end and inner surface of the gown is identified and lifted. The gown is allowed to unroll and drop freely (Fig. 7.2). The hands are inserted into armholes and gown is tied on the back by assistant (Fig. 7.3).

- Sterile pre-powdered gloves are then put on in a way to avoid any contact between skin and outer surface of the glove. The inner surface of right hand glove is grasped with left hand and right hand glove is put on (Fig. 7.4). Then fingers of gloved right hand are inserted inside the folded edge of left glove and the left glove is put on (Fig. 7.5). Double gloves should be used in high-risk patients (HIV +ve, viral hepatitis). The punctured gloves should be replaced immediately.
- The patient's skin in operative area is then cleaned with antiseptic solution (cetrimide, povidone iodine, chlorhexidine). Cleaning should be done systematically from center to periphery so that there is minimum contamination of area of skin incision.
- Sterile drapes are placed to cover all the body except area of skin incision.
- After surgery, all wastes should be disposed of in different bags as per protocol (Fig. 7.6).
- There should be a separate exit for dirty linen and waste to minimize the risk of contamination. It is ideal to have a 'dirty corridor' surrounding the theater complex that carries waste matter in sealed impermeable bags to incinerator machine for destruction.

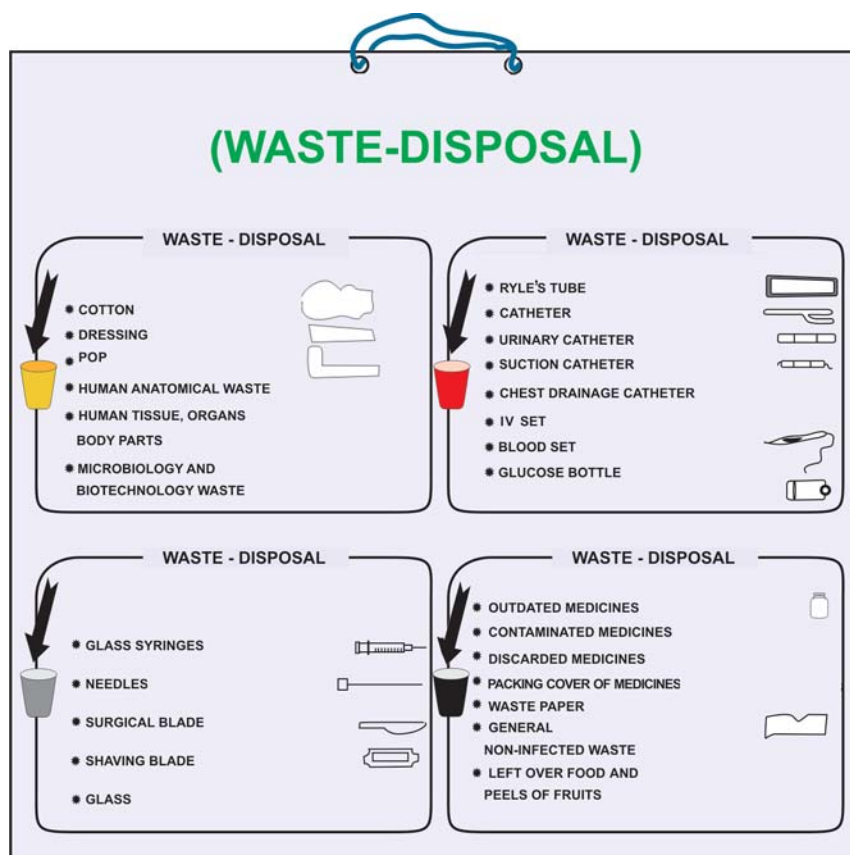


Fig. 7.6: Protocol for waste disposal

STERILIZATION

- *Sterilization* is the process of killing all micro-organisms including bacteria, virus, fungi, parasites and spores.
- *Disinfection* is the process of killing all micro-organisms except spores.
- The first and most important step of disinfection or sterilization is thorough mechanical cleaning of the instruments with soap and water to remove all traces of mucus, pus and blood remains of previous operation.
- Sterilization can be achieved by either *physical* or *chemical agents*.
- The *agents* used for sterilization can be classified in an alphabetic order (Box 7.1):
 - ☐ Autoclaving
 - ☐ Boiling
 - ☐ Chemicals
 - Alcohol
 - Aldehydes

- Aniline dyes
- Halogens
- Phenols and cresols
- Peracetic acid

- ☐ Dry heat
- ☐ Ethylene oxide
- ☐ Formaldehyde
- ☐ Gamma rays

Box 7.1: Sterilization

- Thorough mechanical cleaning of instruments.
- Autoclaving (steam under pressure) is most reliable method.
 - ☐ 15 pounds pressure at 121°C. temperature for 15-45 minutes.
 - ☐ Air tight packing of instruments.
 - ☐ Monitoring done with chemical indicator.
- Gluteraldehyde and Peracetic acid for flexible endoscopes, rubber and plastic equipments.
- Ethylene oxide—for heat sensitive equipments.
- Gamma rays—for commercial use.



Fig. 7.7: Tightly packed instruments put in autoclave chamber

Autoclaving

- It is the most reliable method of sterilization (Box 7.2).
- The *principle* of autoclave is to use steam under pressure. Water normally boils at 100°C. when its vapor pressure becomes equal to atmospheric pressure. When atmospheric pressure is increased in a closed vessel, the water boils at a higher temperature. This steam under pressure has greater power of penetration. It gives up its latent heat by condensing into water and this heat kills all microorganisms including spores.
- *Caution:* All instruments should be packed properly in such a way that no air remains in the autoclave chamber (Fig. 7.7). It is because air is a bad conductor of heat and will decrease the killing power of steam under pressure.
- In autoclaving, 15 pounds pressure is used at temperature of 121°C for 15-45 minutes.

Box 7.2: Autoclaving

- Most reliable method.
- Thorough mechanical cleaning of instruments.
- Temperature of 121°C at 15 lb pressure for 15-45 minutes.
- Latent heat kills the spores.
- Sterilization of instrument, linen, culture media.

Caution

- Air tight packing of instruments.

Box 7.3: Monitoring of efficacy of sterilization

- Impregnated tapes
 - Browne's tubes (chemical indicator)
 - Thermocouples
- If pressure is increased to 30 pounds at temperature of 134°C, autoclaving can be done within 3 minutes. This can sometimes be used in emergency situations where instruments are needed urgently for life saving procedures.
 - Autoclaving is used for sterilizing instruments, dressing materials, gowns, linen and culture media.
 - Monitoring of efficiency of autoclaving is done with chemical indicators or impregnated tapes using spores of *Clostridium tetani* (nontoxic strain) as test organism (Box 7.3).

Boiling

- When instruments are boiled in water (100°C) for 30 minutes, vegetative bacteria are killed but there is no action on spores. So it is not an ideal method for sterilizing instruments.
- However, it can be used in emergency situations in remote areas where facilities for autoclaving are not available.

Chemical Methods

- Alcohol:** Isopropyl alcohol is used for rapid killing of bacteria but has no action on spores. 70% ethanol is used for skin preparation of surgical site, disinfection of thermometers.
- Aldehydes:** 2% aqueous solution of glutaraldehyde (Cidex) is commonly used as disinfectant for endoscopes, rubber and plastic equipments (catheters, endotracheal tubes) and other heat sensitive hospital equipments.

The instruments should be thoroughly rinsed with sterile saline solution before use to prevent irritant effect of the glutaraldehyde solution.

- Aniline dyes:** Acriflavin and proflavin are used as skin and wound antiseptics for dressing.
- Halogens:** Iodine is mainly used as skin disinfectant. Iodophores are organic complexes of iodine and a synthetic detergent. It liberates 1% free iodine that destroys gram +ve as well as gram -ve bacteria but not the spores. It does not stain skin and clothes and is hypoallergic.

It is used in surgical scrubs for hands as well as for operating site.

However, it does not have adequate residual activity.

- e. *Phenols and Cresols*: 1% phenol (carbolic acid) was first used by Lister as skin disinfectant and he was named as 'Father of Antiseptic Surgery'.

Chlorhexidine gluconate (Hibiscrub) is combined with isopropanol and used for surgical hand scrub and skin wound cleanser. Unlike iodophores, it has prolonged residual activity after skin application.

- f. *Peracetic acid (steris)*: It is effective against all micro-organisms including spores. Its mechanism of action is by denaturation of proteins and destruction of cell membrane. It is active even in presence of organic matter. It is particularly useful for sterilization of flexible endoscopes. The system consists of a microprocessor through which chemical fluid constantly circulates at temperature of 50-56°C and the cycle is of 12 minutes.

Dry Heat

- It causes protein denaturation and oxidative damage to the organism. However, it is not effective on spores and is inefficient in comparison to moist heat (autoclaving).
- Dry heat is used in:
Bunsen flame, incineration and hot air oven.
- It is useful in disinfection of sharp and delicate instruments, ointments, grease, oils, glassware and airtight containers.
- It cannot be used for substances like plastic, rubber and intravenous fluids that get denatured.

Ethylene Oxide (ETO)

It is a highly penetrating gas used for killing bacteria as well as spores. It is used in specially designed chambers in which temperature and humidity can be controlled and air can be evacuated. It acts best when applied to clean and dry surfaces. It is used for sterilization of delicate surgical instruments with optical lenses, catheters, tubes, disposable syringes, plastic parts of heart lung machine and ventilators (Fig. 7.8).

Formaldehyde

- Formaldehyde gas is used for fumigation of operation theater, ICU and rooms after treatment of septic cases.



Fig. 7.8: Plastic disposable items sterilized in ETO chamber

- Formaldehyde gas can be combined with dry, saturated steam and it gives sterilization at low temperature (73°C).
- It is suitable for heat sensitive equipments, e.g. flexible endoscopes, cables and plastic materials. It is not useful for airtight equipments.
- Some plastics may absorb formaldehyde and cause allergic reaction on use.

Gamma Irradiation

Gamma rays (from cobalt 60) or high energy electrons (from electron accelerators) are used commercially to sterilize large batches of syringes, catheters, cannulas and surgical blades. It is also known as cold sterilization as it does not lead to rise in temperature. Hence, it is also used for sterilization of heat sensitive pharmaceuticals.

Routinely used methods of sterilization are given in Box 7.4.

Box 7.4: Routinely used methods of sterilization

Equipment	Method
Metal instruments (forceps, retractors, etc.)	Autoclaving
Sharp instrument (scissor, knife, etc.)	Glutaraldehyde
Endoscopes	Peracetic acid
Plastic tubes, syringes and catheters	Ethylene oxide
Operation theaters	Fumigation (formaldehyde gas)
Thermometers	Isopropyl alcohol



8

Chapter

Hemorrhage, Blood Transfusion and Bleeding Disorders

Nisha Marwah, Sanjay Marwah

Hemorrhage can be classified in following ways:

DEPENDING UPON SOURCE OF BLEEDING

External Hemorrhage

When the bleeding is revealed and seen outside, e.g. epistaxis, bleeding from scalp wound, bleeding during surgery.

Internal Hemorrhage

When the bleeding is concealed and not seen outside, e.g. intracranial hematoma.

DEPENDING UPON NATURE OF BLEEDING VESSEL

Arterial Hemorrhage

It is bright red in color. The blood is emitted as a jet with each heartbeat. The bleeding vessel can be identified and secured easily.

Venous Hemorrhage

It is dark red in color. The blood flow is steady and non-pulsatile. If a large vein is injured, e.g. internal jugular vein, there is tremendous blood loss due to low pressure but high flow bleeding. The bleeding is difficult to stop because the vein gets retracted.

Capillary Hemorrhage

It is bright red in color. There is generalized ooze of blood instead of blood flow from definite sites. It can cause serious blood loss in disorders like hemophilia.

DEPENDING UPON TIME OF HEMORRHAGE

Primary Hemorrhage

It occurs at the time of trauma or surgery.

Reactionary Hemorrhage

It occurs within 24 hrs of trauma or operation. In most of the cases, it occurs within 4-6 hrs. due to dislodgement of blood clot or slippage of ligature. The precipitating factors are:

- Rise in blood pressure during recovery from shock.
- Rise in venous pressure due to coughing, vomiting, etc.

Secondary Hemorrhage

It occurs after 7-14 days of trauma or operation. It is due to infection and sloughing of the vessel wall causing moderate to severe bleeding. In most cases, there is a 'warning hemorrhage' in which the dressing gets soaked with fresh blood. It is followed by sudden severe hemorrhage that may prove fatal.

In advanced head and neck cancer, erosion of carotid artery due to ulcerated and infected growth may cause torrential hemorrhage and death (Fig. 8.1).

DEPENDING UPON VOLUME OF BLOOD LOSS

Mild Hemorrhage

When blood loss is less than 500 ml (in adult patient). This much blood loss is compensated by peripheral vasoconstriction. Hence, there are no significant hemodynamic changes seen in the patient.



Fig. 8.1: Fungating carcinoma eroding common carotid artery leading to torrential hemorrhage and death

Moderate Hemorrhage

When blood loss is 500-1000 ml. In such a situation, peripheral vasoconstriction is not sufficient for maintaining circulation. Hence, there are hemodynamic changes in form of tachycardia and hypotension. The extremities feel cold and clammy due to peripheral vasoconstriction.

Severe Hemorrhage

When blood loss is more than one liter. The patient has all the features of moderate hemorrhage due to peripheral vasoconstriction viz., cold clammy skin, thin thready pulse, tachycardia and hypotension. If bleeding continues, then due to splanchnic vasoconstriction, there is decreased renal perfusion leading to oliguria. If not treated, it may lead to acute tubular necrosis and renal failure. If hemorrhage is not controlled, there is decreased cerebral perfusion leading to cerebral anoxia that manifests as irritability, unconsciousness and irregular respiration. In next stage, there is decreased cardiac perfusion leading to cardiac ischemia, cardiac arrhythmia followed by cardiac arrest.

DEPENDING UPON SPEED OF BLOOD LOSS

Acute Hemorrhage

Massive bleeding in a short span of time. It usually occurs after trauma or surgery.

Chronic Hemorrhage

It is slow bleeding that is small in quantity and continues for a long time, e.g. bleeding piles, bleeding peptic ulcer. The blood volume remains normal because blood loss is replaced by plasma. The patient becomes anemic because blood cells are not replaced. Due to anemia, there is tissue hypoxia that is compensated by increased cardiac output. For treatment of such cases, packed red cells should be used instead of whole blood to prevent extra burden on heart that can cause congestive heart failure.

METHODS FOR DETERMINING THE BLOOD LOSS

It is important to measure the volume of lost blood so that blood volume to be replaced can be estimated (Box 8.1A).

Box 8.1A: Measurement of blood loss

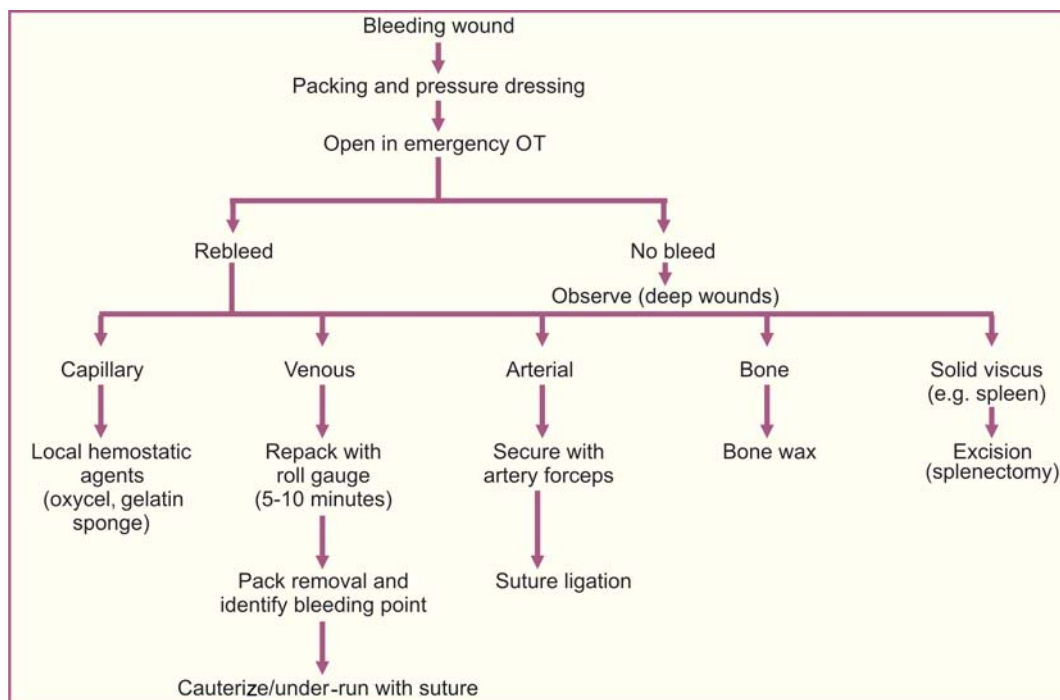
• Size of blood clot	Clot of size of clenched fist = 500 gm.
• Weight of blood soaked swab	Swab weighed before and after surgery. 1 gm increase in weight = 1 ml blood
• Swelling in closed fractures	Closed fracture tibia = 0.5-1.5 liter of blood Closed fracture shaft femur = 1-2 liter of blood
• Fall in hematocrit level	
• Measurement of central venous pressure	

However, estimation of volume of blood loss is difficult and inaccurate because total blood volume is variable at different age. The degree of hemorrhage is now classified into class 1 to class 4 based on estimated blood loss required to produce compensatory changes (Box 8.1B).

Box 8.1B: Classification of hemorrhagic shock

Class	Blood volume lost (%)
1	< 15%
2	15-30%
3	30-40%
4	> 40%

Box 8.2: Control of bleeding



TREATMENT OF HEMORRHAGE

It has two components:

- Control of bleeding
- Restoration of blood volume

Control of Bleeding

This is the prime task and should be done immediately so that further blood loss is minimized (Box 8.2). Various methods are:

Pressure and Packing

Tight packing and pressure dressing is the 'first aid treatment' of any bleeding wound. Any clean and soft linen cloth can be used for this purpose. The packing done on the road side for control of bleeding should always be removed in emergency operation theater. In deep wounds, close observation is required after pack removal even if bleeding appears to have stopped completely.

CASE SUMMARY

18 years female presented with minor scalp injury following road side accident. The patient had severe

bleeding following trauma that was controlled by tight bandage. There was previous history of scalp swelling. In casualty department, the scalp bandage was soaked with blood and resident on duty opened the dressing to examine the wound. As soon as the dressing was opened, the patient bled profusely and lost more than 1 liter of blood and became unconscious. Bleeding could not be stopped with pressure and packing. The patient was rushed to emergency operation theater and was explored under general anesthesia. It was found to be a case of arteriovenous fistula in occipital region that required ligation of feeding vessels. The procedure took more than 4 hours and ten units of blood transfusion to control bleeding.

Learning point: A packed wound with history of severe bleed should always be opened in operation theater.

For bleeding nose due to epistaxis, *digital pressure* using forefinger and thumb can be useful for control of bleeding.

Tourniquet is an elastic pressure bandage applied on the limb away from site of bleeding to control arterial

flow to the bleeding site. These days, it is used occasionally in operation theaters as a prophylactic measure to control bleeding, e.g.

- Limb amputation (peripheral arterial disease is an exception).
- Repair of nerves and tendons, hand surgery.

Position

Raising of the bleeding part above the heart level helps in reducing blood loss by effect of gravity, e.g. bleeding after thyroidectomy is reduced by raising the head end of the bed (reverse Trendelenburg position). Similarly, bleeding from ruptured varicose veins in the legs is reduced by raising the foot end of the bed (Trendelenburg position).

Rest

Sight of blood makes the patient restless and anxious leading to more bleeding. Hence, patient should be well sedated with drugs like pethidine and diazepam.

Operative Methods

During surgery any bleeding point must be controlled except minimal capillary ooze. Large vessels should be clipped with artery forceps taking care not to include surrounding tissues into the bite. This is then dealt with diathermy coagulation or by suture ligation.

If end of a vessel cannot be identified and there is rapid venous bleed, it should be packed with roll gauge for 5-10 minutes. Then on gradual removal of roll gauge, bleeding points are identified and cauterized or under-run with sutures.

If there is generalized slow capillary ooze, it is controlled by applying local hemostatic agents like surgical and abgel.

If there is oozing from bone edges, it is controlled with bone wax.

If a solid viscus is ruptured and bleeds heavily, a part or whole of it may need excision, e.g. splenectomy, nephrectomy, etc.

Restoration of Blood Volume

Withdraw blood sample and send for grouping and cross matching. Start rapid intravenous infusion of a crystalloid solution (Ringer-lactate) using a wide bore cannula (16F). This helps in rapid expansion of

circulatory volume because crystalloid fluid flows easily. The blood transfusion is started as soon as it becomes available. In case, blood is not available or its availability is delayed, various substitutes can be used in its place to buy time (Box 8.3).

Box 8.3: Synthetic substitutes for blood

- | | |
|-----------------------------|---|
| • Dextran | Dextran 40 (low molecular weight)
Dextran 70 (high molecular weight) |
| • Gelatin | |
| • Hydroxyethyl starch (HES) | |
| • Fluorocarbons | |
| • Human albumin (4.5%) | |

BLOOD TRANSFUSION

Indications of blood transfusion are given in Box 8.4.

Box 8.4: Blood transfusion—indications

- | |
|--|
| • Trauma causing severe hemorrhage |
| • Severe burns |
| • Preoperatively, in patients of severe and chronic anemia |
| • Intraoperatively, during major surgery |
| • Postoperatively, in patients who had excessive bleeding during surgery |
| • To arrest bleeding in patients with bleeding disorders (hemophilia) |

Blood Collection

- The donor should be healthy and free from infections like viral hepatitis, AIDS.
- 410 ml blood is drawn from ante-cubital vein.
- Blood is collected in sterile bag containing 75 ml of anticoagulant solution (Acid citrate dextrose).
- Blood is constantly mixed during collection to prevent clotting.

Blood Storage

- Blood is stored in a refrigerator at 4°C.
- It can be stored for three weeks.
- Don't keep blood at room temperature for more than two hours (risk of infection).
- Stored blood has reduced ability to release oxygen.
- Stored blood lacks WBCs and clotting factors (V and VIII) because these are rapidly destroyed.
- Stored blood has reduced platelets.
- If massive transfusion is required, give 1-2 units of fresh blood.

Blood Grouping and Cross Matching

There are two main groups of antigens on human red cells—ABO group and Rh group.

ABO Group

- The red cells contain two antigens A and B.
- The serum contains antibodies namely anti A and anti B.
- On this basis, there are four blood groups (Box 8.5).

Box 8.5: Blood groups

Red cell group (ABO)	Antibody in serum
A	Anti-B antibody
B	Anti-A antibody
AB	No ABO antibody
O	Anti-A and Anti-B antibody
Red cell group (Rh)	
Rh ⁺ ve	D antigen present in red cells
Rh ⁻ ve	D antigen absent in red cells

- For transfusion, red cells of the donor are matched against serum of recipient.
- The person with AB group can receive blood from any body because there is no antibody in serum (universal recipient).
- The person with O group can donate blood to any body because there is no antigen in the red cells (universal donor).

Rh Group

- When red cells contain D antigen, it is called as Rh positive group (seen in 85% of population).
- When red cells lack D antigen, it is Rh negative group (seen in 15% of population).
- If Rh positive blood is given to Rh negative person, anti D antibodies develop against D antigen.
- The first transfusion may be asymptomatic but further transfusion will cause serious incompatibility reactions.
- A similar condition develops when Rh negative mother bears Rh positive fetus.

Transfusion of Blood

- Check compatibility of blood before starting transfusion (Box 8.6).

Box 8.6: Features of compatible blood

- Same ABO group of donor and recipient
- Same Rh group of donor and recipient
- Donor red cells cross match with recipient

- Warm the blood to body temperature before transfusion.
- Start transfusion at a slow rate (5-6 drops/min) for a few minutes and observe for any reaction.
- If there is any doubt of reaction, stop transfusion and give injection frusemide.
- Blood transfusion is done through a filter fitted in BT set that removes small clots and platelet aggregates from stored blood.

Autotransfusion

- Patient's own blood is used for transfusion.
- No risk of transfusion reaction or infection like Hepatitis B and AIDS.
- In elective cases, patient's blood is withdrawn up to 3 weeks before surgery and stored.
- In emergency cases (e.g. ruptured spleen), blood is collected from peritoneal cavity, filtered through autotransfusion apparatus and then transfused.
- Even sterile gauze can be used to filter small clots from collected blood before autotransfusion.

Complications of Blood Transfusion (Box 8.7)

Transfusion Reactions

These may range from mild pyrexial reaction to severe incompatibility.

a. Incompatibility:

- It is due to human errors in collection, labeling and dispatching of blood.

Box 8.7: Complications of blood transfusion

- Transfusion reaction
- Infections
- Coagulation failure
- Congestive heart failure
- Acute renal failure
- Jaundice
- Thrombophlebitis
- Air embolism
- Immunosuppression

- These errors lead to mismatched blood transfusion.
 - The patient complains of
 - ☐ Fever with chills
 - ☐ Nausea and vomiting
 - ☐ Dyspnea
 - ☐ Headache
 - If patient is undergoing surgery under anesthesia, there is sudden hypotension and increased bleeding through wound following mismatched transfusion.
 - In severe cases, there is hemoglobinuria and decreased urine output.
 - Transfusion should be stopped immediately.
 - Intravenous fluid should be rushed along with intravenous frusemide (80-120 mg) to induce diuresis.
 - In extreme cases dialysis may be needed.
 - Sample of patient's venous blood and urine alongwith remaining blood should be sent to blood bank for rechecking.
- b. *Allergic reactions:*
- These are due to allergy to plasma products in donor blood.
 - There is tachycardia and skin rash.
 - Stop transfusion and give antihistaminics (chlorpheniramine 10 mg).
- c. *Pyrexial reactions:*
- These are due to "pyrogens" in the blood or in the transfusion apparatus.
 - These can be avoided by use of disposable plastic sets.
 - The patient develops fever with chills and tachycardia.
 - Stop transfusion temporarily and give antipyretics (paracetamol) and antihistaminics.
 - Once symptoms subside, start transfusion again at a slow rate using fresh disposable transfusion set.
- d. *Sensitization to leukocytes and platelets:*
- It is seen in patients getting multiple transfusions (e.g. thalassemia).
 - The patient develops antibodies against transfused platelets and leukocytes.
 - It can be prevented by giving packed cells.
 - Treatment is antipyretics, antihistaminics and steroids.

Infections

Various infections transmitted by blood transfusion are:

- Serum hepatitis
- AIDS
- Malaria
- Bacterial infection

These infections can only be prevented by proper screening of the donor.

Coagulation Failure

- It usually occurs following massive transfusion or incompatible blood transfusion.
- There is excessive bleeding through wounds, petechial hemorrhages, hematuria, hematemesis, melena, etc.
- Treatment is by replacement of clotting factors with FFP, cryoprecipitate and platelet concentrate.

Congestive Heart Failure

- It usually follows rapid transfusion in patients with chronic anemia.
- It can be avoided by
 - ☐ Giving slow transfusion.
 - ☐ Giving packed cells.
 - ☐ Giving diuretics.

Immunosuppression

Blood transfusion has shown to depress the immune response of the patient. Hence, blood transfusion should be avoided unless clearly indicated.

Problems of Massive Transfusion

Massive transfusion is defined as replacement of patient's whole blood volume with stored RBCs in 24 hrs or transfusion of more than 10 units within a few hours. It can cause following problems:

- Hypothermia
- Acid-base imbalance (metabolic alkalosis)
- Hyperkalemia
- Citrate toxicity (Hypocalcemia)
- Coagulation failure.

Fractions of Blood

In certain conditions, fractions of blood are more useful than transfusing whole blood. These fractions are:

1. *Packed red cells*: Useful in patients with chronic anemia and in elderly patients with poor cardiac reserve.
2. *Platelet rich plasma (PRP)*: Useful in patients with thrombocytopenia. It is prepared by slow centrifugation of fresh donated blood.
3. *Platelet concentrate*: It is prepared by centrifugation of platelet rich plasma. It is also useful in patients with thrombocytopenia. If stored frozen, it remains effective for many months.
4. *Fresh frozen plasma (FFP)*: Plasma is removed from fresh blood and is rapidly frozen and stored at -40°C . It preserves all coagulation factors and is useful in treatment of coagulopathies (hemophilia).
5. *Cryoprecipitate*: When FFP is allowed to thaw at 4°C and supernatant plasma is removed, the remaining cryoprecipitate is rich source of factor VIII. It is stored at -40°C and is used for treatment of patients with hemophilia.
6. *Fibrinogen*: It is prepared from plasma and stored in dried form. It is used for treatment of congenital afibrinogenemia and disseminated intravascular coagulation.
7. *Human albumin*: It is rich in protein and due to heat treatment; it is free from risk of viral hepatitis. It is useful as plasma expander, e.g. in severe burns.

BLEEDING DISORDERS

In a patient scheduled for elective surgery, accurate history and physical examination is important source of information regarding risk of bleeding during operation.

Investigations for Bleeding Disorders

- Bleeding time—for platelet function.
- Clotting time—for clotting factors.
- Prothrombin time (PT)—detects deficiency of clotting factors.
- Activated partial prothrombin time (aPTT)—prolonged in anticoagulant therapy, hemophilia.
- Platelet count.
- Serum fibrinogen levels.
- Thromboelastography (TEG)—it provides numerical and graphic representation of coagulation. It tells both hypocoagulability as well as hypercoagulability states.

Acquired Bleeding Disorders

These are more common than congenital disorders. Various causes are:

Vitamin K Deficiency

- It is due to
 - ☐ Inadequate dietary intake
 - ☐ Obstructive jaundice
 - ☐ Antibiotics
- Treatment is injection Vitamin K 10 mg I/M daily for three days.
- FFP transfusion rapidly corrects the deficiency.

Anticoagulant Drugs

- These should be stopped or neutralized before surgery.
- Oral anticoagulants (warfarin) are neutralized by injection Vitamin K.
- Heparin is neutralized by injection protamine sulphate.

Hepatic Failure

It leads to defective synthesis of clotting factors.

Renal Failure

It causes bleeding disorders due to platelet dysfunction.

Thrombocytopenia

- It presents with petechial hemorrhages, purpura, mucosal bleeding and excessive bleeding during surgery.
- Common causes are drugs and hypersplenism.
- Transfusion of platelet concentrate raises platelet count.

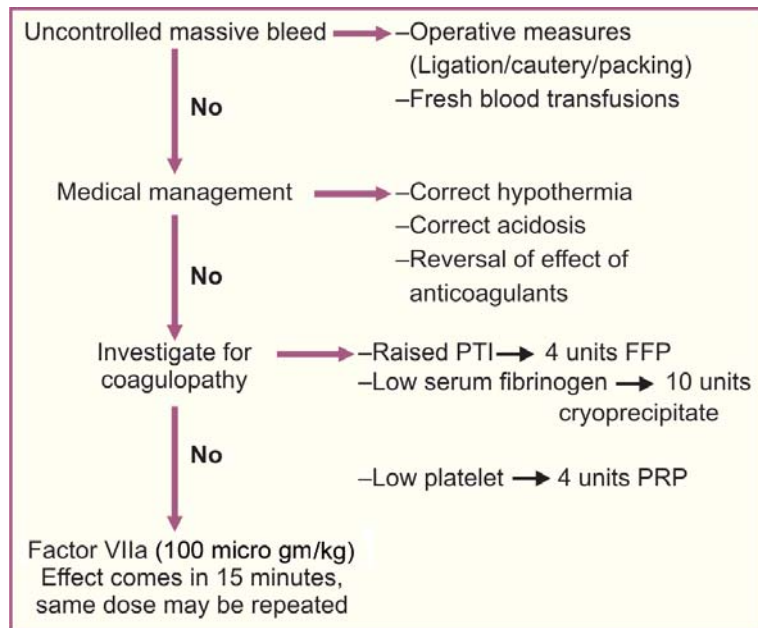
Hypothermia

- It usually occurs following massive transfusion and causes altered coagulation.
- Treatment is rewarming of patient.

Congenital Bleeding Disorders

Hemophilia

- It is X-linked genetic disorder of coagulation.

Box 8.8: Outlines for management of uncontrolled bleeding

- It has two types:
 - ☐ *Hemophilia A*: Due to deficiency of factor VIII.
 - ☐ *Hemophilia B (Christmas disease)*: Due to deficiency of factor IX.
- It almost exclusively affects males while females are carriers.
- When level of coagulation factor in blood is less than 2%, there is severe bleeding.

Clinical features

- ☐ Recurrent bleeding in joints.
- ☐ Epistaxis.
- ☐ Bleeding gums.
- ☐ Bleeding after tooth extraction.
- ☐ Intracranial bleed (may cause death).

Investigations

- ☐ Factor VIII levels are decreased in hemophilia A and factor IX levels are decreased in hemophilia B.
- ☐ aPTT is prolonged and PT is normal.

Treatment

- ☐ In hemophilia A, cryoprecipitate is given that is rich source of factor VIII.
- ☐ In hemophilia B, treatment is platelet concentrate

that contains factor IX along with other clotting factors.

von Willebrand's Disease

- Most common congenital clotting disorder.
- It affects both sexes.
- There is defective platelet function and low factor VIII levels.
- It is due to defect or deficiency of vWF
- Bleeding encountered is similar to bleeding due to platelet dysfunction, e.g. mucosal bleeding, epistaxis, petechial hemorrhages.

Investigations

- ☐ aPTT is prolonged.
- ☐ Bleeding time is prolonged with normal PT.
- ☐ vWF levels and factor VIII levels are decreased.

Treatment

- ☐ Administration of DDAVP (synthetic vasopressin) shortens the bleeding time and normalizes factor VIII and vWF activities.
- ☐ Cryoprecipitate infusion replaces vWF and controls or prevents bleeding.

In recent years, factor VIIa has been found to control bleeding effectively in coagulation defects (Box 8.8).

9 Chapter

Shock, Water-Electrolyte and Acid Base Balance

Sanjay Marwah, Jasbinder Kaur

SHOCK

Definition

It is a clinical syndrome characterized by severe dysfunction of vital organs due to inadequate tissue perfusion.

Pathophysiology

Whenever there is hypotension, immediate vasoconstriction occurs in an attempt to maintain perfusion to vital

organs, viz. brain, heart, kidneys, liver and lungs. Metabolic effects of prolonged hypotension are shown in Box 9.1. If hypotension remains uncorrected and splanchnic vasoconstriction persists, it produces adverse effects on abdominal viscera (Box 9.2). If hypotension and inadequate tissue perfusion still persists, it leads to irreversible shock causing damage of vital organs and death (Box 9.3).

Types of Shock

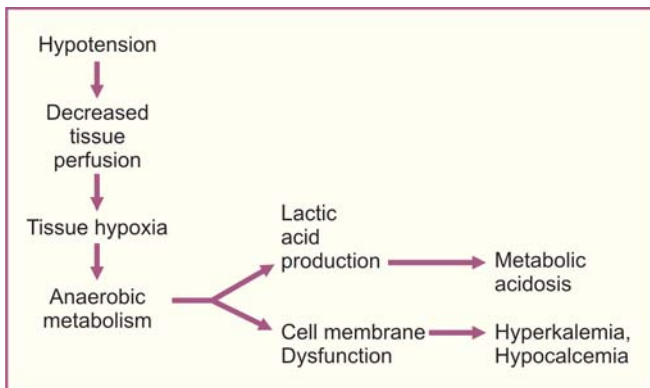
Hypovolemic Shock

It is due to loss of intravascular volume. The causes can be:

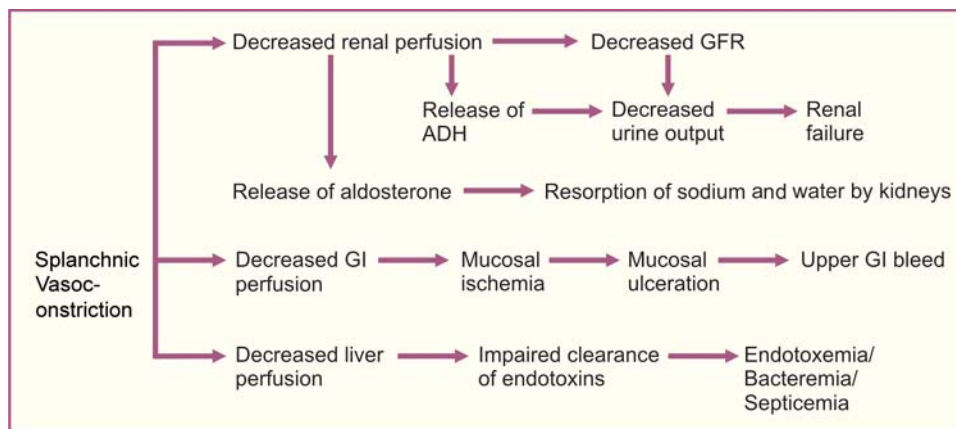
- Hemorrhage
- Dehydration due to vomiting and diarrhea.
- Burns causing loss of plasma.

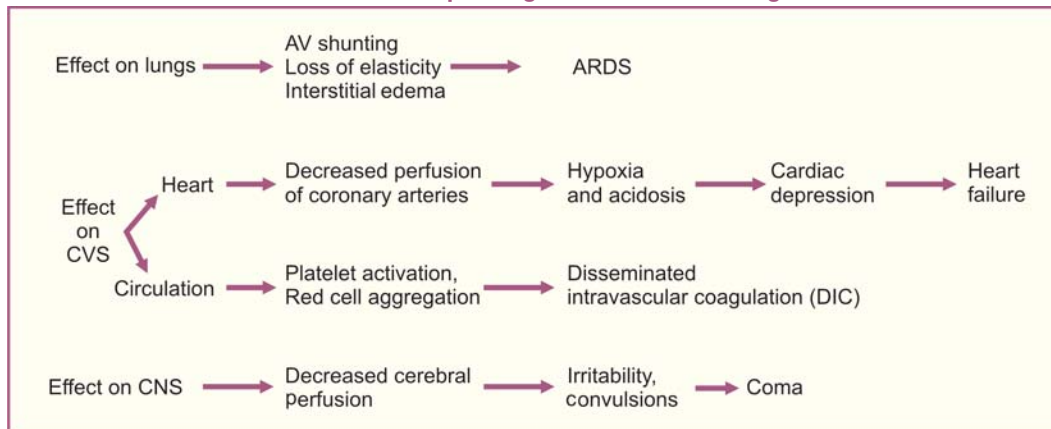
Hypovolemic shock can be further categorized into mild, moderate and severe shock depending upon degree of loss of intravascular volume (Box 9.4).

Box 9.1: Pathophysiology of shock



Box 9.2: Effects of splanchnic vasoconstriction



Box 9.3: Effect of prolonged shock on vital organs**Box 9.4: Types of hypovolemic shock**

Parameters	Mild	Moderate	Severe
Volume loss	< 1 liter	1-2 liter	>2 liter
Symptoms	Asymptomatic	Mild anxiety, restlessness, thirst, sweating	Severe anxiety, disorientation, air hunger, cold extremities
Pulse	70-80/min (N)	90-100/min	>120/min
BP (Systolic)	120 mm Hg (N)	90-100 mm Hg	< 70 mm Hg
CVP	5-10 cm H ₂ O (N)	0-5 cm H ₂ O	Minus value (very low)
Urine output	60 ml/hr (N)	<30 ml/hr	Nil
(N: Normal)			

Neurogenic Shock

It is caused by sympathetic failure leading to vasodilatation, peripheral pooling of blood and hypotension. It leads to reduced cerebral perfusion, cerebral hypoxia and unconsciousness. Various causes are:

- Injury to spinal cord causing paraplegia.
- Spinal anesthesia.
- Vasovagal shock that follows severe pain, e.g. dental extraction without effective local anesthesia.

The patient should be made to lie down immediately with raised feet (Trendelenburg position) to improve cerebral perfusion. If patient is kept propped upright, it may lead to irreversible brain damage and/or cardiac arrest leading to death. If hypotension persists, rapid intravenous fluids and vasopressors should be given.

Cardiogenic Shock

It is due to failure of pump mechanism of heart. Various causes are:

- Massive myocardial infarction.
- Pulmonary embolism causing blockade of pulmonary vessels and right ventricular failure.
- Cardiac compression from outside viz. pericardial effusion.

In right ventricular failure, there is engorgement of neck veins, liver enlargement and edema feet. In left ventricular failure, crepitations appear in the base of lungs.

Anaphylactic Shock

It is due to hypersensitivity to a drug, toxin or serum leading to acute circulatory collapse.

The clinical features are:

- Skin rash.
- Difficulty in breathing due to bronchospasm and laryngeal edema.
- Sudden hypotension.
- Loss of consciousness.

Septic Shock

It is due to infection caused by bacteria, virus, fungi or protozoa. In majority of cases, it is due to gram-negative sepsis and common infecting organisms are *E. coli*, *Klasiella*, *Proteus*, *Pseudomonas* and *Bacteroids*. Clinically, there are two types of septic shock:

Early warm shock Toxins cause cutaneous vasodilatation and skin becomes warm and pink. The patient has fever with chills. There is tachycardia and hypotension. Diagnosis is easily made since there is associated focus of infection in the body, e.g. paratonsillar abscess, carbuncle etc.

Late cold shock If toxemia persists, it leads to increased capillary permeability, hypovolemia, decreased cardiac output, tachycardia and vasoconstriction. The skin becomes cold and clammy. The patient becomes drowsy and tachypneic. Clinically it becomes difficult to differentiate from hypovolemic shock; the only guide is presence of septic focus. If toxemia still remains uncontrolled, it can lead to multiple organ dysfunction syndrome (MODS) and multiple system organ failure (MSOF) (See Chapter 3: Infections).

Treatment of Shock

General Measures

- Establishment of clear airway and maintenance of adequate ventilation and oxygenation.
- In case patient is unconscious with breathing difficulty, endotracheal intubation with ventilatory support may be required.
- Maintenance of blood pressure (systolic BP > 90 mm Hg).
 - A large-bore cannula (16G) is inserted into forearm vein and isotonic fluid (Ringer lactate or normal saline) is rapidly infused. In case of blood loss, it is replaced by blood transfusion.
 - Inotropic drugs (Dopamine, Dobutamine) cause vasoconstriction and improve myocardial

Box 9.5: Monitoring of patient in shock

- Urine output
- Blood pressure
- Pulse oximetry
- ECG
- CVP
- Blood gas analysis

contractility. These may be required as I/V infusion if hypotension persists.

- However in cardiogenic shock, restriction of fluid is required.
- The adequacy or inadequacy of fluid replacement is best judged by *Central venous pressure (CVP) monitoring* (Box 9.5). A 20 cm long intravenous catheter is passed into right subclavian vein or right internal jugular vein with patient in supine and head down position. The catheter tip is advanced up to superior vena cava (Fig. 9.1). Intravenous saline infusion is connected to the catheter. Before starting infusion, aspirate with a syringe to check the back flow of blood into the catheter to confirm patency and presence of cannula in the vein. The tubing of infusion line is connected to saline manometer through a stopcock. A reference point marking the position of right atrium is taken as “zero”. This zero

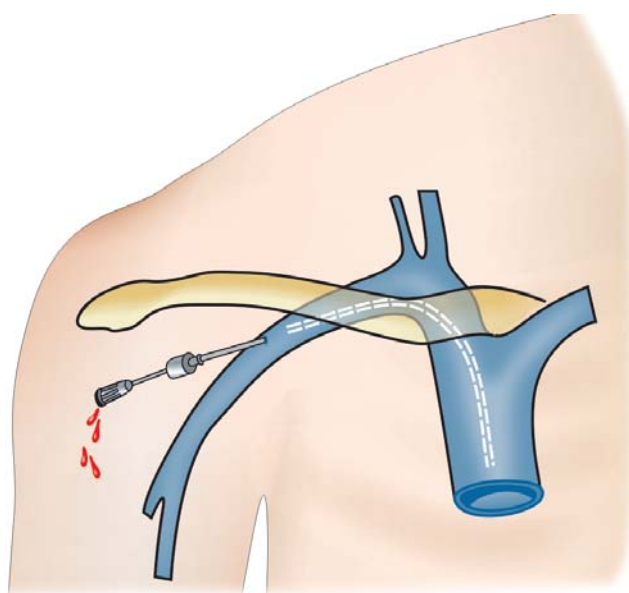


Fig. 9.1: Cannula inserted in right subclavian vein below and parallel to medial one-third of clavicle

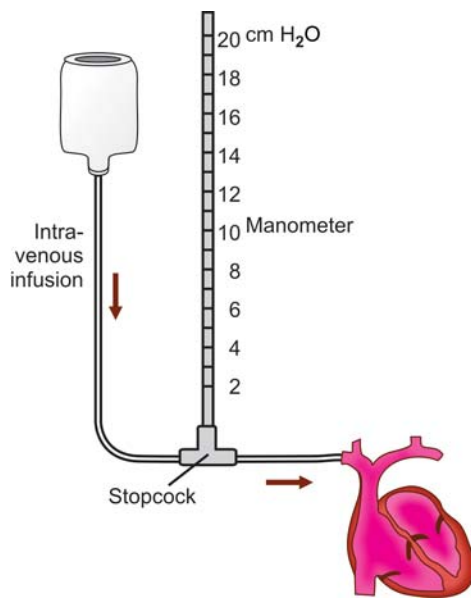


Fig. 9.2: Stopcock (zero marking) connecting infusion line to manometer

marking on the manometer should begin at the level of stopcock that is kept at the level of patient's midaxillary line (Fig. 9.2).

If CVP is low, intravenous fluid supplement should be given (e.g. hypovolemic shock). But if CVP is normal or raised, I/V fluids should be restricted (e.g. cardiogenic shock). Complications of central venous cannulation are given in Box 9.6.

Box 9.6: Complications of central venous cannulation

- Pneumothorax
- Hemothorax
- Arrhythmia
- Air embolism
- Brachial plexus injury
- Infection

- **Pulmonary capillary wedge pressure (PCWP)** is a better device to monitor left ventricular function and cardiac output. A balloon tip catheter (Swan-Ganz catheter) is introduced into right atrium. The balloon is inflated with 1.5 ml air and advanced via right ventricle into pulmonary artery while monitoring pressure tracing. The tracing becomes flat when balloon gets wedged

Box 9.7: Indications of PCWP

- Cardiogenic shock (better differentiation of left and right heart failure)
- Septic shock
- Pulmonary arterial hypertension
- Monitoring of fluid administration

Box 9.8: Complications of PCWP

- Pulmonary infarction
- Pulmonary artery rupture
- Cardiac arrhythmia

into a small branch to give capillary pressure. When the balloon is deflated, it gives pulmonary artery pressure (PAP).

CVP reflects only right atrial pressure while PCWP tells left ventricular pressure and is better method for monitoring cardiogenic shock (Box 9.7 and 9.8).

- *Catheterization* of urinary bladder and monitoring of urine output. If urine output is more than 30 ml/hr, it indicates adequate renal perfusion.
- *Correction of metabolic acidosis:* It is estimated by serial arterial blood gas analysis and corrected by I/V sodium bicarbonate.

Specific Measures

Hypovolemic shock

- Fluid replacement.
- In hemorrhagic shock, control of bleeding and blood replacement (See Chapter 8: Hemorrhage, Blood Transfusion and Bleeding Disorders).

Anaphylactic shock

- Maintenance of airway.
- Injection Hydrocortisone 200-400 mg I/V.
- Injection adrenaline 0.5 mg S/C, I/M or I/V.
- Vasopressors (dopamine, mephentine) for hypotension.
- Bronchodilators (Injection deriphylline, aminophylline) for bronchospasm.

Septic shock

- Treatment of infection by:
 - Appropriate antibiotics.
 - Surgical drainage/debridement of wound (See Chapter 3: Infections).

- Treatment of shock by:
 - ☐ I/V fluid infusion.
 - ☐ Vasopressor drugs.
 - ☐ Steroids in high doses over a short period are protective against endotoxemia. Single dose of methyl prednisolone (15-30 mg/kg) is given I/V and repeated after 4 hrs. It improves cardiac, renal and pulmonary functions and does not impair immune response of the body.

WATER AND ELECTROLYTE BALANCE AND IMBALANCE

The input and output of water and electrolytes are finely balanced in the body.

The **daily input** of water is derived from two sources (Box 9.9):

- **Exogenous** in form of liquid intake and ingested solid food. The solids consumed contribute to the half of water requirement.
- **Endogenous** is released from oxidation of ingested food.

The **daily output** of water is by four routes (Box 9.9):

- **Urine**—daily output of urine is about 1500 ml/day. Minimum 30 ml/hr urine is required to excrete the toxic metabolites from the body.
- **Faeces**—about 100 ml/day water is lost through this route normally.
- **Lungs**—about 400 ml/day water is lost in expired air from the lungs.
- **Skin**—about one liter water is lost daily through skin as perspiration meant for thermoregulation. The loss occurring through skin and lungs is called **insensible loss**.

This regulation is mainly done by the hormones:

- ADH (Antidiuretic hormone) secreted in response to rise in plasma osmolality that causes increased reabsorption of water in the distal renal tubules.

Box 9.9: Daily input output balance in an adult

Input		Output	
Liquids	1200 ml	Urine	1500 ml
Solids	1000 ml	Skin	1000 ml
Oxidation of food	300 ml	Lungs	400 ml
		Faeces	100 ml

- Aldosterone—produced by the *zona glomerulosa* of the adrenal cortex.
- Renin-angiotensin mechanism—releases renin by the juxtaglomerular cells in response to decrease in renal plasma flow.

Osmolality: It is the osmotic pressure exerted by the number of moles per kg of solvent. Important electrolytes which determine osmotic pressure of our body fluids are Na^+ , K^+ , Cl^- and HCO_3^- . K^+ is the most important electrolyte of **intracellular fluid** while Na^+ , Cl^- and HCO_3^- are important for **extracellular fluid**. Commonly carried out investigations show the status of ECF. Osmolality of plasma varies between 285-295 mOsm/kg.

Disturbances in Water Balance

- Hypovolemia
- Hypervolemia.

Hypovolemia

It is due to diminished water intake (pure water depletion).

Causes

- Decreased water intake—due to inability to swallow, e.g. painful ulcers in oral cavity, esophageal obstruction.
- Excess loss of water—loss from gut, e.g. vomiting, diarrhea.
 - ☐ Insensible loss from skin and lungs, e.g. fever
 - ☐ Loss from lungs, e.g. after tracheostomy.

Clinical features The patient complains of thirst, weakness and decreased urine output.

Investigations Raised hematocrit, increased specific gravity of urine, increased blood urea, increased serum sodium.

Treatment

- If swallowing is possible, increase oral intake of water.
- If there is difficulty in swallowing or in case of severe hypovolemia, give intravenous 5% dextrose or dextrose saline.

Hypervolemia

It is due to excess intake of water orally or excess infusion of fluids parenterally.

Causes

- Rapid and excess infusion of IV fluids
- Water retention enema
- Fluid retention due to cardiac or renal failure
- Excess absorption of fluid from prostatic fossa during transurethral resection of prostate
- ADH secreting tumor, e.g. oat cell tumor of lung.

Clinical features

- Nausea, vomiting, drowsiness, weakness, convulsions and coma.
- Patient passes large amount of dilute urine.
- Although patient appears to be in shock, but on examination, pulse and blood pressure normal, neck veins distended, pedal edema.

Investigations Low hematocrit, blood urea normal, serum sodium may be low.

Treatment

- Restrict water intake.
- Very slow intravenous infusion of hypertonic saline.

Disturbances in Electrolyte Balance

Four important disorders are:

- Hybernatriemia
- Hyponatriemia
- Hyperkalemia
- Hypokalemia.

Hypernatremia

It is the sodium excess in body (more than 150 mmol/l).

Causes

- Mismanaged fluid administration (excess saline in postoperative period)
- Mineralocorticoid excess.

Clinical features Puffiness of face, pitting edema, weight gain, distended jugular veins. Pulmonary edema may occur in neglected cases.

Treatment Water administration orally or through Ryle's tube, 5% dextrose IV

Hyponatremia

It is the sodium depletion in body (less than 135 mmol/l).

Causes

- Excess vomiting or Ryle's tube aspiration causing loss of intestinal secretions.
- Intestinal fistula.

- Severe diarrhea.
- Postoperative hyponatremia—it is due to prolonged administration of sodium free solutions (5% dextrose) intravenously.
- Syndrome of inappropriate anti-diuretic hormone secretion (SIADH)—it is due to excess ADH secretion following surgery or trauma, more often seen in elderly patients. Excess ADH causes water retention and increase in ECF volume. This in turn leads to decreased aldosterone secretion and excess loss of sodium in urine.
- Pseudohyponatremia—serum osmolality depends on various solutes like sodium, glucose, urea, plasma lipids and proteins. Out of these, sodium is most abundant and others have less concentration. However, when their concentration becomes very high, the relative concentration of sodium becomes less. So despite normal concentration, the serum sodium levels become less and it is termed as pseudohyponatremia.

Clinical features

- Unlike hypovolemia, thirst is not evident in hyponatremia
- Sunken eyes
- Drawn face
- Dry, coated tongue
- Dry and wrinkled skin
- Collapsed peripheral veins
- Low blood pressure
- Urine is small in amount and dark colored.

Investigations

- Hematocrit increased
- Serum sodium decreased
- Urine sodium decreased (In SIADH urine sodium increased)
- Urine specific gravity high.

Treatment

- Treat underlying cause.
- IV infusion of isotonic saline or Ringer's lactate.

Hyperkalemia**Causes**

- Excessive K⁺ intake with diuretics (K⁺ sparing)
- Parenteral infusion of K⁺
- Transfusion of stored blood
- Acute renal failure (oliguric phase)

- Acidosis
- Addison's disease
- Tissue damage (hypoxia, severe dehydration, hemolysis)
- Catabolic states (diabetes)
- Fallacious values because of hemolysed sample/contamination.

Clinical features

- Vague muscle weakness
- Flaccid paralysis
- In severe cases (K^+ levels > 10 mmol/L), there can be ventricular fibrillation and death.

Investigations

- Serum K^+ levels > 5.5 mmol/L
- ECG changes—Tall, peaked T-wave followed by absence of P-wave and finally formation of abnormal QRS complex.

Treatment

- Glucose and insulin to promote influx of K^+ in cells.
- 10 ml of 10% of calcium gluconate IV.
- Retention enema.
- If above mentioned measures fail, peritoneal or hemodialysis is helpful.
- Treatment of the cause.

Hypokalemia

Causes

- Diuretics
- Parenteral nutrition
- Diuretic phase of acute renal tubular necrosis and chronic renal failure.

- Renal tubular acidosis
- Alkalosis
- Mineralocorticoid excess
- Severe trauma
- Major surgical operation (increased ADH and aldosterone)
- Anabolic states
- Chronic diarrhea
- Excessive use of purgatives
- Intestinal fistulae
- Insulin administration.

Clinical features

- Muscle weakness
- Weakness of respiratory muscles causing rapid, shallow, gaping breathing
- Abdominal distention due to paralytic ileus
- Cardiac arrhythmias / congestive cardiac failure.

Investigations

- Serum K^+ levels < 3.5 mmol/L (decreased serum K^+ indicates much larger depletion of K^+)
- ECG changes—depressed ST segment, low or inverted T-wave.

Treatment

- Dietary intake in mild cases (common foods have enough K^+).
- K^+ salts / I V KCl (Slow drip) in moderate to severe cases. Urine output should be adequate.
- Treatment of the cause.

Comparison between hyperkalemia and hypokalemia is given in Box 9.10.

Box 9.10: Comparison between hyperkalemia and hypokalemia

	<i>Hyperkalemia</i>	<i>Hypokalemia</i>
Clinical features	Flaccid paralysis Ventricular fibrillation and death	Muscle weakness Abdominal distention Cardiac arrhythmias / congestive cardiac failure
K^+ levels	> 5.5 mmol/L	< 3.5 mmol/L
ECG changes	Tall, peaked T-wave followed by absence of P-wave and finally formation of abnormal QRS complex.	Depressed ST segment, low T-wave
Treatment	Glucose and insulin to promote influx of K^+ in cells. 10 ml of 10% of calcium gluconate IV. If above mentioned fails, peritoneal or hemodialysis. Treatment of the cause.	Dietary intake in mild cases. K^+ salts / IV (Slow drip) in moderate to severe cases. Treatment of the cause.

Postoperative Fluid Therapy

Period of Therapy

First 24 hours: Due to stress of operative trauma, adrenal steroids (aldosterone) and ADH are released in circulation resulting in retention of sodium and water and excretion of potassium from the kidneys (**Sodium stays, potassium flees**). The requirement of sodium and water is thus reduced. Moreover, due to body reserves of potassium, its replacement is also not required in first 24 hours.

In a healthy adult, approximately 2 liters of fluid (500 ml N saline and 1500 ml 5% dextrose) is required during first 24 hours.

After 24 hours: The fluid requirement after 24 hours is calculated by measuring previous days' urine output and adding it to insensible loss from skin and breathing. In case, there is some additional loss, e.g. due to fever, diarrhea, Ryle's tube aspirate, etc. then it is also taken into account. For example:

Insensible loss	1000 ml
Urine output	<u>1500 ml</u>
Total output	2500 ml

Thus, in a patient kept nil orally, replacement of 2500 ml IV fluids (equal to previous days' output) is required during next 24 hours. It comes out to be 5 bottles of 500 ml each.

The daily need of sodium is 100 mmol and potassium is 40-60 mmol.

Thus requirement will be met by giving one bottle (500 ml) of N saline, four bottles (500 ml each) of 5% dextrose and two ampoules (20 ml each) of KCL added to the infusion bottle. The potassium supplement should not be given as IV bolus as it can cause arrhythmia.

In case of electrolyte imbalance, serum levels of sodium and potassium will guide for calculating the requirements.

Once patient starts taking orally, the IV fluid supplement is decreased accordingly.

Types of IV fluids

Types of fluids used for IV use are:

- Crystalloids
- Colloids

Crystalloids These are solutions of electrolytes in water. They are available as bottles containing sterile, pyrogen free solution without preservative and for single IV infusion. Routinely used solutions are:

1. **5% dextrose:** It is isotonic solution that supplies calories without electrolytes. It is useful in early post-operative period when sodium excretion is reduced. Its prolonged administration can lead to hyponatremia.

A bottle contains 500 ml solution of dextrose is in the strength of 5% w/v.

Each 100 ml contains:

- Dextrose—5 gm
- Water for injection in QS
- Calories 17 kcal/100 ml

- 2 **Isotonic saline (0.9%) solution:** It is needed as replacement fluid when large amount of sodium has been lost, e.g. by vomiting, Ryle's tube aspiration, intestinal fistula, etc.

Its other uses are:

- To dilute and dissolve drugs
- As irrigating fluid
- To toilet the body cavity
- Treatment of alkalosis (Hypochloremic) with dehydration
- Treatment of mild hyponatremia.

In a bottle of 500 ml, each 100 ml contains:

- Sodium chloride—0.9 gm
- Water for injection in QS.

- 3 **Dextrose-saline solution:** It contains 4.3% dextrose and 0.18% saline and is isotonic (5% dextrose in saline is hypertonic). It is also used as maintenance/replacement fluid.

4. **Ringer's lactate solution:** It contains sodium, potassium and chloride in almost same concentration as that of plasma. It also contains some calcium and bicarbonate as lactate. It does not contain dextrose. It is ideal replacement fluid in hemorrhagic shock due to trauma, surgery, etc. while awaiting blood (poor man's white blood).

Contraindication to its use are:

- Liver disease, severe hypoxia and shock where lactate metabolism is impaired and lactic acidosis may occur due to infusion of Ringer's lactate solution.
- Severe metabolic acidosis where conversion from lactate to bicarbonate is impaired. So it can worsen acidosis.

5. Other fluids used are:

- **Isolyte P:** It is designed to suit maintenance fluid requirement of children (more water and less electrolytes).

- *Isolyte G*: It is gastric replacement solution and is used to replace loss of gastric juice (in vomiting, Ryle's tube aspiration) and in treatment of metabolic alkalosis.
- *Isolyte M*: It is ideal fluid for maintenance therapy.
- *Isolyte E*: It is used as extracellular replacement solution. It has electrolytes similar to ECF except double amount of potassium and acetate which will get converted into bicarbonate. It should be avoided in metabolic alkalosis.

Colloids These are fluids having substances of high molecular weight like proteins, starch or gelatin dissolved in water, efficient to produce oncotic pressure. They can be:

- Natural—albumin and plasma protein fractions.
- Synthetic/artificial—Dextran, Gelatin (Haemaccel), Hydroxy ethyl Starch (HES).

Synthetic colloids are preferred due to following advantages:

- Easily available
- Economic
- No transmission of diseases
- Low incidence of anaphylactic reactions.

The features of ideal colloid are given in Box 9.11.

Box 9.11: Features of ideal colloid

- Rapid replacement of blood loss
- Sustained hemodynamic parameters
- Sufficient long intravascular life
- Produces sufficient oncotic pressure
- Improve oxygen supply
- Improve organ functions by perfusion
- No transmission of disease
- Easily metabolized and excreted
- No effects on cross-matching of blood
- No effect on coagulation
- No anaphylactic or allergic reaction

1. *Gelatins (Hemaccel)*: It contains polymer of degraded gelatin with electrolytes. Its intravascular stay time is 2-3 hr and it gives oncotic pressure of 21 mm Hg.

Its indication/uses are:

- As plasma expander in hypovolemic shock, burns, trauma.
- Perioperatively to replace blood loss.
- As preloading fluid in spinal anesthesia.

Dose 20 ml/kg/day (1000ml / 50 kg).

Contraindications

- Allergy to gelatin solutions
- State of fluid overload.

Side effects

- Anaphylactic /allergic reactions (0.146%).

Demerits

- Colloid osmotic pressure low (21)
- Water binding capacity low (15 ml/gm)
- Short stay in vascular compartment (2-3 hr)
- May interfere with coagulation.

2. *Hydroxy ethyl Starch (HES)*: It is isotonic colloid derived from maize and is used as replacement fluid. It is composed of amylopectin derived from starch. Its preparations are:

- *HES-200 (Pentastarch)* molecular weight 200,000
- *HES-450 (Hetastarch)* molecular weight 450,000
- *Tetrahes-130 (Tetrastarch)* molecular weight 130,000

All preparations are in N saline.

Indications/Uses

- As plasma expander in hypovolemia, trauma, surgery
- Preloading in spinal anesthesia
- Hemodilution in cardiac and vascular surgeries
- Improves tissue perfusion and oxygen utilization in shock.

Contraindication

- Allergy to salt
- Fluid overload.

Side effects

- Allergic reactions
- Bleeding disorders.

Precautions

- HES may increase the renal toxicity of aminoglycoside antibiotics.
- Interference with blood grouping and cross matching.
- Rapid infusion may cause circulatory disturbances and subsequent damage to tissues. So infusion should be given slowly.

Dose

- 20 ml/kg/hr for adult.

3. **Dextran 40, 70:** It is a polysaccharide (glucose polymer) synthesized by fermentation of sucrose that is ultimately degraded enzymatically to glucose.

Each 100 ml Dextran 40 contains:

Dextran-40—10 gm

Sodium chloride—0.9 gm

Water for injection—QS

Its water binding capacity is 25 ml/gm and osmotic pressure is 290 mOsm/L.

Dose – 20 ml/kg/day

Intravascular stay period

Dextran – 40 (10%)—2-4 hr

Dextran – 70 (6%)—6 hr

Uses

- As plasma expander
- As antithrombotic agent
- To improve perfusion in vascular surgeries (Dextran 40).

Contraindications

- Allergy to Dextran
- Overhydration
- Coagulation disorder

Side effects

- Interfere with cross-matching due to rouleaux formation
- Increased bleeding time due to decreased platelet adhesiveness
- Anaphylactic reaction
- Noncardiogenic pulmonary edema (Direct toxic effect on pulmonary vasculature).

4. **Human albumin:** It is derived from pooled human plasma and is a costly preparation.

100 ml of 25% solution contains 25 gm albumin and half life of albumin is 16 hr.

Water binding capacity is 17 ml/gm of albumin.

Indications

- When crystalloids fail to sustain plasma volume for more than a few minutes because of low oncotic pressure.
- Abnormal loss of protein from vascular space as in peritonitis and burns.

Contraindication

- Allergy to albumin and fluid overload.

Comparison between crystalloid and colloid is given in Box 9.12.

Box 9.12: Comparison between crystalloid and colloid

	<i>Crystalloid</i>	<i>Colloid</i>
Composition	Water + electrolytes	High mol wt substance
Pressure	Osmotic pressure	Oncotic pressure
Distribution	Extravascular space	Intravascular space
Volume requirement	3 times of loss	Equal to loss
Cross-matching	No effect	Interfere
Cause edema	Yes	No
Anaphylaxis	No	Do occur
Cost	Economic	Costly

ACID BASE BALANCE AND DISORDERS

Concept of pH

- pH of a solution is defined as the negative logarithm of the hydrogen ion concentration. As it is “negative log”, so pH decreases as H^+ concentration increases. Normal pH of blood is 7.4 (range 7.36-7.44).
- A unit change in pH means 10 times change in hydrogen ion concentration. Hence, although pH change appears small, it is sufficiently large in terms of H^+ concentration.
- A **buffer** is a mixture of a weak acid and its conjugate base or salt. The buffers maintain the pH of body fluids within normal limits.
- **K_A** is called dissociation constant of the acid and it tells about degree of dissociation (strength) of the acid. Strong acids are completely dissociated. Therefore, larger the value of K_A , more dissociated or stronger the acid.
- **Henderson Hasselbalch equation** relates pH of buffer solution to pK_A of its weak acid and the ratio of molar concentration of the weak acid and its salt.

$$pH = pK_A + \log \frac{[\text{Base/salt}]}{\text{Acid}}$$

- When a strong acid is added to a buffer solution it reacts with the salt part of the buffer pair. This neutralizes the added acid generating an equivalent amount of the buffer acid. In this way a strong acid is replaced by a weak acid and pH is maintained.

- Different acids generated in body can be divided into three groups.
 - **Carbonic acid:** It is formed by hydration of CO_2 .
 - **Fixed acids:** The examples are H_2SO_4 and H_3PO_4 . Since these acids are not volatile, hence called as fixed acids.
 - **Organic acids:** The examples are lactic acid, acetoacetic acid, β -hydroxy butyric acid, uric acid, etc.
- A large change in pH is not compatible with proper functioning of tissues. A proper pH is necessary for structural and functional integrity of proteins (including enzymes), nucleic acids and membranes. A large change in pH alters ionization of certain groups of amino acids (and proteins), purine and pyrimidine bases and certain components of phospholipids. Concentration of certain free ions like Ca^{++} depends on pH of our body fluids. These free ions are important in regulation of excitability of excitable tissues like muscle and nervous tissue.
- Buffers form the first line of defense against incoming acids or alkalis. A useful buffer should keep pH of body fluids close to 7.4, should be present in high concentration and the pKa value of its weak acid should be close to 7.4.
- Important buffers of the body are:
 - Hemoglobin and protein buffers
 - Phosphate buffer
 - Bicarbonate buffer
- **The bicarbonate buffer** is most important buffer of the body. It has bicarbonate (HCO_3) and carbonic acid (H_2CO_3) as two components and their normal ratio is 20 : 1. Alteration in this ratio alters the pH regardless of absolute values of HCO_3 and H_2CO_3 . A decrease in ratio leads to acidosis while increase leads to alkalosis. The bicarbonate level can be altered by metabolic factors while carbonic acid level is regulated by respiratory factors. Alteration in one is automatically followed by compensation by the other thus maintaining their ratio and therefore pH of blood tends to remain constant. The excess of H_2CO_3 is eliminated as CO_2 by lungs while HCO_3 is regulated by the kidneys.

Acid Base Disorders

Acid base disorders are classified according to changes in components of bicarbonate-carbonic acid buffer, since

these can be easily evaluated. The three components (pH, HCO_3 and pCO_2) of this buffer are related as follows (the Henderson-Hasselbalch equation):

$$\begin{aligned}\text{pH} &= \text{pK} + \log [\text{HCO}_3] / [\text{H}_2\text{CO}_3] \\ &= \text{pK} + \log [\text{HCO}_3] / \text{pCO}_2 \\ &\text{as } [\text{H}_2\text{CO}_3] \text{ can be replaced by } \text{pCO}_2.\end{aligned}$$

Whenever there is disturbance in acid base balance in the body, the changes are labeled as primary disorders. In order to correct these changes and to normalize the pH, certain compensatory changes occur (Box 9.13).

Box 9.13: Compensatory changes in acid base disorders

Primary disorder	Primary abnormality	Compensation
Metabolic acidosis	$\downarrow \text{HCO}_3 \rightarrow \downarrow \text{pH}$	Respiratory ($\downarrow \text{pCO}_2$)
Metabolic alkalosis	$\uparrow \text{HCO}_3 \rightarrow \uparrow \text{pH}$	Respiratory ($\uparrow \text{pCO}_2$)
Respiratory acidosis	$\uparrow \text{pCO}_2 \rightarrow \downarrow \text{pH}$	Renal ($\uparrow \text{HCO}_3$)
Respiratory alkalosis	$\downarrow \text{pCO}_2 \rightarrow \uparrow \text{pH}$	Renal ($\downarrow \text{HCO}_3$)

Compensation in Acid Base Disorders

Respiratory regulation:

- Respiratory regulation is important in **metabolic acidosis and alkalosis**.
- In metabolic acidosis, because of decrease in bicarbonate, the ratio $\text{HCO}_3/\text{H}_2\text{CO}_3$ is reduced and accordingly pH is reduced. This stimulates chemoreceptors and causes reflex hyperventilation leading to CO_2 wash-off. This reduces H_2CO_3 and tends to normalize pH. It may however be pointed out that although ratio is normalized, the actual concentrations of both HCO_3 and H_2CO_3 are reduced. These concentrations are then normalized by the renal regulatory processes.
- In metabolic alkalosis the ratio $\text{HCO}_3/\text{H}_2\text{CO}_3$ is increased because of increase of HCO_3 . pH is, accordingly, increased. This reduces chemoreceptor stimulation, resulting in hypoventilation and consequent CO_2 retention. This increases H_2CO_3 thereby tending to normalize the $\text{HCO}_3/\text{H}_2\text{CO}_3$ ratio. This tends to normalize pH, although, the actual HCO_3 and H_2CO_3 concentrations are both increased. These concentrations are then normalized by the renal regulatory processes.

- It should be remembered that the pulmonary response in normalization of the ratio $\text{HCO}_3/\text{H}_2\text{CO}_3$ is incomplete and therefore, pH is not completely normalized. This is because the effect of pH in respiratory response is opposed by the prevailing pCO_2 . For example, in acidosis reduced pH stimulates respiration while reduced pCO_2 opposes the response. Similarly in alkalosis the raised pH depresses respiration but increase in pCO_2 tends to stimulate respiration.
- In metabolic acidosis and alkalosis the **pulmonary compensation is rapid** and uncompensated cases are not seen. For example, in metabolic acidosis one will always find reduced HCO_3 (primary disorder) and reduced pCO_2 or reduced H_2CO_3 (pulmonary compensation). Similarly in metabolic alkalosis one will find increased HCO_3 (primary disorder) and increased H_2CO_3 or increased pCO_2 (pulmonary compensation).

Renal regulation:

- Renal regulation is important both in **metabolic acid base disorders** as well as **respiratory acid base disorders**.
- In respiratory acidosis the ratio $\text{HCO}_3/\text{H}_2\text{CO}_3$ is reduced because of retention of CO_2 and increase of H_2CO_3 . To normalize pH renal excretion of HCO_3 is reduced and generation of new HCO_3 is increased. This will normalize $\text{HCO}_3/\text{H}_2\text{CO}_3$ ratio, although, the actual amounts of both the components are increased. These can only be normalized by removal of primary pulmonary disorder.
- Similarly in respiratory alkalosis the ratio $\text{HCO}_3/\text{H}_2\text{CO}_3$ is increased because of excessive loss of CO_2 (and reduction of H_2CO_3). To normalize pH, renal excretion of HCO_3 is increased and generation of new HCO_3 reduced. pH is thus normalized by restoration of $\text{HCO}_3/\text{H}_2\text{CO}_3$ ratio, although, the actual amounts of both components are reduced. The actual amount can not be normalized unless the causative pulmonary disorder is treated.
- In respiratory acid base disorders the renal compensation is a slow process and therefore both uncompensated (**acute disorder**) and compensated (**chronic disorder**) cases are seen. In acute cases of respiratory acidosis one may find increased pCO_2 (or H_2CO_3) and normal HCO_3 while in chronic cases both the components are increased. Similarly

in acute respiratory alkalosis only pCO_2 (H_2CO_3) is reduced while in chronic cases both the components are reduced. It may also be realized that normalization of pH in respiratory disorders will only occur when the slow renal response has produced the desired effect on HCO_3 component of the buffer.

There are four primary acid base disorders:

- Metabolic acidosis
- Metabolic alkalosis
- Respiratory acidosis
- Respiratory alkalosis.

Metabolic Acidosis

- It is a condition in which there is deficit of base or excess of any acid other than carbonic acid.
- Primary change is $\downarrow \text{HCO}_3$ or $\uparrow \text{H}^+ \rightarrow \downarrow \text{pH}$
- For each $\downarrow$ in HCO_3 of 7-7.5 mmol/L, pH $\downarrow$ by 0.1
- Compensatory change is $\downarrow \text{pCO}_2$, H^+ excretion in urine (acidic urine).
- Expected pCO_2 in metabolic acidosis = $1.5 \times \text{HCO}_3 + 8 (\pm 2)$.

Causes

- Increase in fixed acid
 - ☐ Ketoacidosis in diabetes, starvation
 - ☐ Lactic acidosis due to tissue hypoxia and anaerobic metabolism in hypovolemia, septic shock, cardiac arrest, etc.
 - ☐ Renal failure
 - ☐ Salicylate poisoning.
- Loss of base
 - ☐ Prolonged Ryle's tube aspiration
 - ☐ High intestinal fistula
 - ☐ Ulcerative colitis
 - ☐ Prolonged diarrhea.

Clinical features

- Rapid, deep, noisy respiration due to stimulation of respiratory centers (**Kussmaul's respiration**).
- Tachycardia and hypotension in patients of septicemia.
- Central nervous system depression (fatigue, confusion, stupor).
- Oliguria with strongly acidic urine.

BGA report

- $\downarrow \text{pH}$
- $\downarrow \text{HCO}_3$

- A typical BGA report will be as follows:

Metabolic acidosis	pH 7.3	pCO ₂ 20	HCO ₃ 9
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Treatment

- To correct hypoxia, restore adequate tissue perfusion by rapid infusion of ringer lactate solution.
- Administration of sodabcarb solution should not be done routinely (Box 9.14).
- Sodabcarb should only be given in cases of severe acidosis (pH < 7.2) or cases with base deficit.
- Calculating dose of HCO₃:
 - 1 ml sodabcarb (7.5%) contains 0.9 mmol of HCO₃.
 - HCO₃ requirement (mmol/lit) = 0.3 × body weight (kg) × base deficit (mmol/lit).
 - Initially give only half of the required dose of sodabcarb IV slowly and repeat only if required based on blood pH value.

Box 9.14: Harmful effects of excessive and rapid HCO₃ administration

1. Hypokalemic cardiac toxicity if patient is K⁺ depleted
2. Tetany in a patient of renal failure or having hypocalcemia
3. Congestive heart failure or worsening of hypertension due to excessive intake of Na⁺
4. In acidosis there is hyperventilation as respiratory center (RC) is stimulated (from acid pH of both blood and cerebrospinal fluid). As plasma HCO₃ is corrected RC shall remain stimulated as CSF HCO₃ does not quickly equilibrate with plasma HCO₃. It may result in respiratory alkalosis

Anion Gap

- There are unmeasured anions in blood (proteins, PO₄⁻, SO₄⁻) = 23 mmol/L.
- There are unmeasured cations in blood (Ca⁺⁺, K⁺, Mg⁺⁺) = 11 mmol/L.
- The anions are more than cations and the difference is called anion gap. The normal anion gap = 12-18 mmol/L.
- When organic acids increase (lactic acid, ketoacids), there is increase in anion gap (>20 mmol/L)
- Anion gap is used for evaluation of patients with metabolic acidosis.
- Accumulation of H⁺ (e.g. lactic acidosis) leads to high anion gap.

- Anion gap remains unchanged in cases of metabolic acidosis due to loss of HCO₃ ions (e.g. intestinal obstruction, intestinal fistula) because lost HCO₃ is replaced with chloride ions (hyperchloremic acidosis).
- This helps in diagnosis of cause of acidosis. In most of the cases, however, careful history may be enough and study of the anion gap may not be required.

Metabolic Alkalosis

- It is a condition in which there is excess of base or deficit of any acid other than carbonic acid.
- The alkalosis due to loss of acid is almost always associated with hypokalemia.
- Primary change is ↑ HCO₃ or ↓ H⁺ → ↑ pH.
- For each ↑ in HCO₃ of 7-7.5 mEq/L-pH ↑ by 0.1.
- Respiratory compensation
 - ↑ pCO₂
 - ↑ HCO₃ excretion by kidneys (alkaline urine)
- Expected pCO₂ in metabolic alkalosis = 0.7 × HCO₃ + 21 (± 2).

Causes—two types

- a. Chloride responsive
 - Loss of acid from stomach, e.g. vomiting, prolonged Ryle's tube aspiration
 - Volume depletion (Chloride losing diarrhea)
 - Diuretics (long-term use)
- b. Chloride nonresponsive
 - Potassium depletion (low serum K)
 - Diuretics (recent use)
 - Corticoid excess (over administration, Cushing's disease).

BGA report

- ↑ pH
- ↑ HCO₃
- A typical BGA report will be as follows:

Metabolic alkalosis	pH 7.55	pCO ₂ 50	HCO ₃ 42
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Clinical features

- Cheyne-Stokes' respiration with apnoic spells (cessation of breathing) of 5-30 sec.
- Tetany.
- Associated features of hypokalemia, e.g. lethargy, muscle weakness.

Treatment

- Saline infusion for chloride responsive.

- Chloride deficit (mEq/L) = $0.3 \times \text{wt. (in kg)} \times (100 - \text{Plasma chloride})$
- Volume of isotonic saline (L) = Chloride deficit/154.

- For chloride nonresponsive—correct hypokalemia, correct corticoid excess.

Respiratory Acidosis

- It is a condition in which pCO_2 is above normal range.
- Primary change is $\uparrow \text{pCO}_2 \rightarrow \downarrow \text{pH}$
- For each 10 mm Hg $\uparrow \text{pCO}_2$ – pH $\downarrow$ by 0.05
- Compensatory change is $\uparrow \text{HCO}_3$.
 - Acute respiratory acidosis: For each 10 mm Hg $\uparrow \text{pCO}_2$, $\text{HCO}_3 \uparrow$ by 1 mEq/L.
 - Chronic respiratory acidosis: For each 10 mm Hg $\uparrow \text{pCO}_2$, $\text{HCO}_3 \uparrow$ by 3 mEq/L.

Causes

- Inadequate ventilation of anesthetized patient.
- Incomplete reversal of muscle relaxants at extubation following general anesthesia.
- Surgery in patients with underlying lung disease, e.g. COPD, severe acute asthma.
- Others (fever, anxiety, hyperthyroidism, pulmonary edema, cirrhosis).

BGA report

- $\downarrow \text{pH}$
- $\uparrow \text{pCO}_2$
- A typical BGA report will be as follows:

Respiratory acidosis	pH 7.1	pCO ₂ 90	HCO ₃ 30
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Clinical features: The features are primarily of underlying problem.

Treatment

- Correction of underlying pathology.
- Oxygenation.
- Ventilatory support.

Respiratory Alkalosis

- It is a condition in which pCO_2 is below normal range.
- Primary change is $\downarrow \text{pCO}_2 \rightarrow \uparrow \text{pH}$.
- For each 10 mm Hg $\downarrow \text{pCO}_2$ – pH $\uparrow$ by 0.1.
- Compensatory change is $\downarrow \text{HCO}_3$ by increased renal excretion of HCO_3 .

- Acute respiratory alkalosis: For each 10 mm Hg $\downarrow \text{pCO}_2$, $\text{HCO}_3 \downarrow$ by 2 mEq/L.
- Chronic respiratory alkalosis: For each 10 mm Hg $\downarrow \text{pCO}_2$, $\text{HCO}_3 \downarrow$ by 4-5 mEq/L.

Causes

- Hyperventilation under anesthesia
- Hyperpyrexia
- Head injury (Hypothalamic lesion)
- High altitude
- Hysteria
- Anxiety
- Sepsis.

Clinical features

- The features are primarily of underlying problem.
- During anesthesia, alkalosis is accompanied with pallor and fall of BP.

BGA report

- $\uparrow \text{pH}$
- $\downarrow \text{PCO}_2$
- A typical BGA report will be as follows:

Respiratory alkalosis	pH 7.55	pCO ₂ 20	HCO ₃ 22
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Treatment CO₂ rebreathing.

How to Read an ABG Report ?

An arterial blood sample is taken from the femoral or radial artery and subjected to blood gas analysis. The acid base disorders can be recognized by interpreting the ABG (Arterial Blood Gas) report (Box 9.15).

Box 9.15: Normal ABG report

pH	:	7.40 (7.35-7.45)
pO ₂	:	80-104 mm Hg
pCO ₂	:	40 mm Hg (35-45)
HCO ₃	:	24 + 2 mEq / L
BE	:	0 + 2
O ₂ sat	:	96-98%
Na	:	135-148 mEq / L
K	:	3.5-5.5 mEq / L
Ca	:	1.13-1.32 mEq / L
Cl	:	98-106 mEq / L
Anion gap	:	12 mmol / L

- **pH** tells about H⁺ in the blood.

- **pO₂** is measurement of partial pressure of oxygen in blood.
- **pCO₂** is measurement of partial pressure of CO₂ in blood.
- **HCO₃** (standard bicarbonate) is concentration of serum bicarbonate after fully oxygenated blood has been equilibrated with CO₂ at 40 mm Hg.
- **BE** (Base excess or deficit) is total of buffer anions present in the blood in excess or deficit of normal. Base excess or deficit multiplied by 0.3 of body weight in kg gives the total extracellular excess or deficit of base in mmol.
- **Anion gap** is discussed above.

Calculating Acid Base Status from ABG Report:

Step 1: First look at pH

- ↓ pH (<7.35)—acidosis
- ↑ pH (>7.45)—alkalosis
- Normal pH (7.35-7.45).

Step 2: Look at pCO₂

- pH ↓ and pCO₂ ↑ = Primary Respiratory Acidosis
- pH ↑ and pCO₂ ↓ = Primary Respiratory Alkalosis.

An easy way to remember is that if change in pH and pCO₂ occurs in opposite directions (one increasing and other decreasing), the problem is respiratory.

Step 3: Look at HCO₃

- pH ↓ and HCO₃ ↓ = Primary Metabolic Acidosis
- pH ↑ and HCO₃ ↑ = Primary Metabolic Alkalosis

An easy way to remember is that if change in pH and HCO₃ occurs in same directions (both increasing or both decreasing), the problem is metabolic.

If both PaCO₂ and HCO₃ are out of normal range and pH is also out of range, such a disturbance is called **Mixed disorder**.

Step 4: Study compensation

- **In Metabolic Disorders**, the respiratory compensation causing retention or removal of CO₂ occurs in few minutes to few hours. Calculate difference between measured and expected pCO₂ using formulae given in Box 9.16.
- If measured pCO₂ is greater than the expected pCO₂, it implies that the respiratory system is not compensating for the metabolic acidosis and respiratory acidosis is also present.

Box 9.16: Formulae for evaluation of compensation in metabolic disorders

Metabolic disorder	Expected pCO ₂
Metabolic acidosis	$1.5 \times \text{HCO}_3 + 8 (+2)$
Metabolic alkalosis	$0.7 \times \text{HCO}_3 + 21 (+2)$

- **In respiratory disorders**, first determine change in pH and HCO₃ to decide whether it is acute or chronic problem. Then calculate difference between measured and expected pH using formulae given in Box 9.17

Box 9.17: Formulae for evaluation of compensation in respiratory disorders

Respiratory disorder	Expected pH
Respiratory acidosis	
Acute	$7.4 - [(\text{observed pCO}_2 - 40) \times 0.008]$
Chronic	$7.4 - [(\text{observed pCO}_2 - 40) \times 0.003]$
Respiratory alkalosis	
Acute	$7.4 + [(40 - \text{observed pCO}_2) \times 0.008]$
Chronic	$7.4 + [(40 - \text{observed pCO}_2) \times 0.001]$

Step 5: Anion gap estimation

If metabolic acidosis is diagnosed—check anion gap to find the cause of acidosis.

Step 6: Assessment of oxygenation

- The value of pO₂ depends upon inspired oxygen concentration (FiO₂).
- The expected pO₂ of a person can be estimated with the formula:
Expected pO₂ = FiO₂ % × 5
For example, if a person is given 25% oxygen, his expected pO₂ is 25 × 5 = 125 mm Hg
- pO₂ < 80 mm Hg is hypoxemia.
- pO₂ < 60 mm Hg is life threatening.
- The relation between pO₂ and FiO₂ is given in Box 9.18.

Box 9.18: Relation between pO₂ and FiO₂

Clinical condition	pO ₂ / FiO ₂
Normal	> 5
Some oxygenation problem	3-5
Acute lung injury	2-3
ARDS	< 2

10

Chapter

Care of the Acutely Injured

Sanjay Marwah

- Trauma is the leading cause of death during young age (30-40 years) when person is in most productive period of his life.
- 40% of trauma deaths can be avoided by preventive measures.
- **Trimodal distribution** of trauma deaths:
 - i. *First peak*: Death occurs at the time of injury. It is due to injury to major organs like brain, heart and great vessels. Primary prevention is the only way to reduce these deaths.
 - ii. *Second peak*: Death occurs several hours after injury. The period between second and first peak is called as the 'golden hour'. Deaths during 'golden hour' are caused by airway, breathing and circulatory problems and most of these are potentially treatable conditions.
 - iii. *Third peak*: Death occurs days or weeks after injury. The cause of death is infection and organ failure. Proper initial management on admission can reduce morbidity and mortality during this period.

PREHOSPITAL MANAGEMENT AND FIRST AID OF TRAUMA PATIENTS

It has three components (Box 10.1).

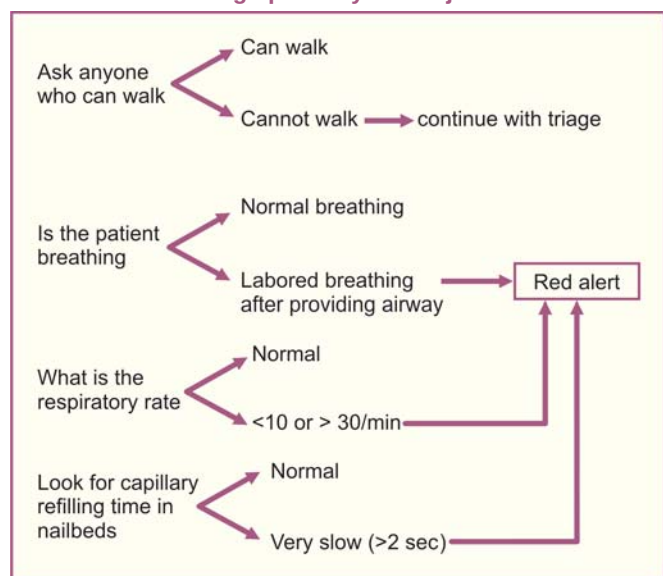
Box 10.1: Prehospital care of severely injured patient

- Triage
- Treatment
- Transport

Triage

The term triage literally means "to sort". In a mass casualty, the goal of prehospital triage is to identify high-

Box 10.2: Triage pathway for major accidents



risk injured patients. They are at maximum risk of dying from their injuries and thus would benefit from management at a trauma center. An outline of pathway to be followed in a major trauma event is given in Box 10.2.

Treatment

- Is victim breathing?—**No**—Provide airway and give mouth-to-mouth breathing.
- Is there pulse or heartbeat?—**No**—Do external cardiac massage.
- Is there gross external bleeding?—**Yes**—Elevate the part and apply external pressure to stop bleeding.
- Is there any possibility of injury to spine?—**Yes**—Protect neck and spine of the patient before moving him. For patient shifting, three or four persons lift



Figs 10.1A and B: Log rolling the patient

the patient straight without any movement of spine (**log rolling** the patient) (Figs 10.1A and B).

- Is there any fracture of long bones?—**Yes**—Do splinting.

Transport

The transport of critically injured patient is ideally done in ground ambulance equipped with life saving equipments like oxygen cylinder, ventilator, power points, infusion pumps, emergency drugs, etc. If ambulance is not available, a truck or wagon is preferred over a passenger car. It is because loading the patient in passenger car needs manipulations that may be more harmful than waiting for proper transport. The patient should be transported in supine position escorted by a doctor or trained paramedic and resuscitation should continue during the transport. Ideally, the transport time should not exceed 15-30 minutes. For long distance transport and in hilly terrain, transport by aircraft is an

ideal mean, but it carries a high cost and is not available at most places. On reaching hospital, the important information required to be handed over by accompanying person is MIST (Box 10.3).

Box 10.3: Important prehospital information

M—Mechanism of injury
I—Injuries sustained or suspected
S—Signs (vital signs on scene and during transport)
T—Treatment given (drugs, fluids, etc.)

IN HOSPITAL MANAGEMENT OF TRAUMA PATIENTS

The Advanced Trauma Life Support (ATLS) system developed by American College of Surgeons' Committee on Trauma focuses primarily on the first hour of trauma management and aims at reduction in preventable deaths. The ATLS manual provides following guidelines for management of acutely injured patient:

PRIMARY SURVEY

It is a rapid and systematic evaluation to detect and manage life-threatening injuries (Box 10.4). A trauma team should be there to manage airway, breathing and circulation problems simultaneously.

Box 10.4: Primary survey

A—**Airway** and total spine control
B—**Breathing** and ventilatory support
C—**Circulation** with hemorrhage control
D—**Disability** (brief neurological evaluation)
E—**Exposure** (completely undress the patient)

A. Airway

- The first priority in a critically injured patient is to establish and maintain a patent airway. It helps in delivering sufficient oxygen to tissues and avoids hypoxemic organ damage.
- Management of airway should always be combined with control of cervical spine (with hands/lateral blocks/hard cervical collar).
- Ask the patient his name. If he can answer, his airway is intact and he has adequate cerebral perfusion.
- Initial assessment of airway is done by: **Look, Listen** and **Feel** (Box 10.5).

Box 10.5: Airway assessment

Look	Cyanosis
	Chest movements
	Respiratory rate
	Trauma (Maxillofacial, chin, mouth, neck, chest)
Listen	Voice quality
	Breath sounds
	Abnormal sounds (crepts, rhonchi)
Feel (with hands)	Chest movements
	Subcutaneous emphysema
	Tracheal position
	Broken teeth/ foreign bodies in oral cavity
	Tongue fall

- In case of acute airway obstruction, management is done as follows (Box 10.6):
 - ☐ High flow oxygen is administered by face mask.
 - ☐ *Head tilt*: Flexing the cervical spine and then extending the head backwards improves airway patency.
 - ☐ *Chin lift* combined with opening the mouth clears the tongue fall.
 - ☐ *Jaw thrust*: The mandible and tongue are displaced anteriorly by pushing forwards the angle of the mandible. However, it can cause significant movement of an unstable cervical spine.
 - ☐ *Oropharyngeal or nasopharyngeal airway* (Fig. 10.2) can be used to improve a partially obstructed airway. However, a nasal airway is inappropriate in suspected fracture base of skull.



Fig. 10.2: (A) Face mask, (B) oropharyngeal and (C) nasopharyngeal airway

Box 10.6: Management of airway

Patient can talk	High flow oxygen, control cervical spine
Patient unconscious, noisy breathing	Oral suction, chin lift, head tilt, Try oropharyngeal/ nasopharyngeal airway
Unable to obtain clear airway (maxillofacial injury, bleeding, vomiting, burns)	Endotracheal intubation
Failed intubation	Cricothyroidotomy/Tracheostomy

- If patient still has labored breathing or no breathing (apneic), it is an indication for urgent endotracheal intubation. Other indications for tracheal intubation are given in Box 10.7.

Box 10.7: Indications for endotracheal intubation

Immediate: Apnea

Urgent: Inadequate breathing after jaw thrust and airway insertion
Depressed level of consciousness (GCS<8)
Risk of aspiration pneumonia (from vomitus, blood)

- As an alternative to endotracheal intubation, laryngeal mask airway (LMA) (Fig. 10.3) can be used for ventilation.
- If endotracheal intubation fails, surgical cricothyroidotomy or tracheostomy may be performed under local anaesthesia.

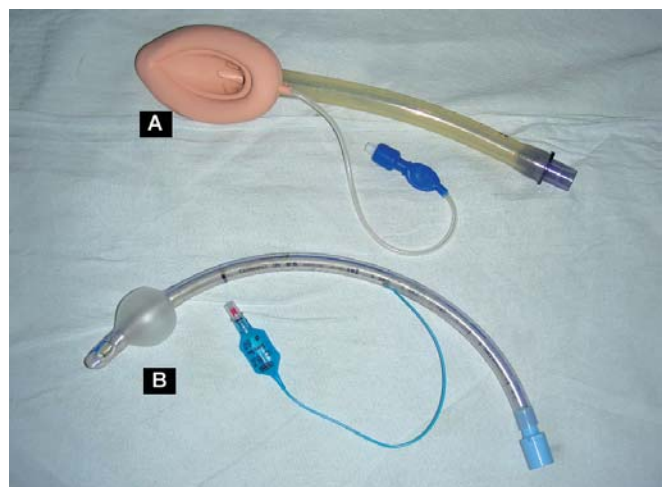


Fig. 10.3: (A) Laryngeal mask airway and (B) cuffed endotracheal tube

a. *Surgical cricothyroidotomy*: It is a life saving procedure and must be done quickly as hypoxic brain injury occurs within 3-5 minutes of no oxygenation.

Surgical anatomy: The cricothyroid membrane is an avascular fibroelastic membrane between thyroid cartilage (above) and cricoid cartilage (below). The laryngeal prominence or “Adam’s apple” is the most important landmark. Identify the cricothyroid membrane by feeling a notch inferior to laryngeal prominence.

Equipments:

Size 10 scalpel blade.

Size 6-7 tracheostomy tube.

Tracheal spreader or artery forceps.

Procedure:

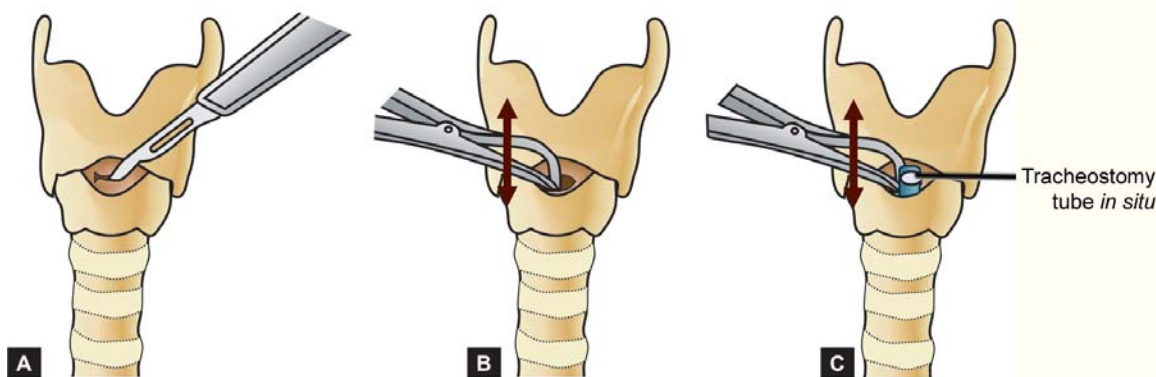
- Patient is placed in supine position.
- Locate cricothyroid membrane by palpating Adam’s apple and moving downwards.
- Give local anesthesia (if there is time and patient is conscious).
- Stabilize the thyroid cartilage with left hand. Make a horizontal stab incision in midline through skin and cricothyroid membrane allowing only tip of the scalpel blade to enter trachea. Enlarge the opening using artery forceps or tracheal spreader (Figs 10.4A to C). Insert a cuffed tube into the hole directing the tube distally into the trachea and inflate the cuff. Attach a connector to the tube and ventilate the patient.

b. *Needle cricothyroidotomy*: It can be done as an emergency life saving measure when equipment for cricothyroidotomy is not available. A large bore (12-14 G) cannula is introduced through the skin and

cricothyroid membrane in downward direction to enter the trachea. High flow oxygen is given through the cannula. Arrange for a definitive airway as needle cricothyroidotomy is only suitable as a temporary measure (10-15 minutes).

c. *Tracheostomy*: (See Chapter 16: Diseases of Larynx).

- *In fractures of facial skeleton*, edema develops within 60-90 minutes. Thus, immediately after injury to facial skeleton, airway might appear to be adequate. But it gets occluded rapidly by swelling of the tongue, facial and pharyngeal tissues causing acute respiratory obstruction. Hence, oropharyngeal airway should be inserted during initial period (golden hour) even if airway appears to be unobstructed. If it is not done, an emergency tracheostomy may be required later with risk of hypoxic damage.
- *Massive nasopharyngeal bleeding causing airway obstruction*: In case of severe facial hemorrhage following trauma, it can prove fatal without early recognition and definitive first aid. The patient presents with bleeding from nose and mouth that is staining cloths, bed, on the attendants and on the floor. Attempt to CT scan these patients without control of bleeding may result in death by exsanguination. The correct steps in management are:
 - Secure the airway by cricothyroidotomy/tracheostomy and ventilate.
 - Pass two 22F Foley’s catheters through two nostrils and hook by index finger into the mouth.
 - A roll gauze pack is secured through eye of each catheter with 0 nylon stitch and wedged in post-



Figs 10.4A to C: Surgical cricothyroidotomy

nasal space using digital pressure and traction on catheters.

- ❑ The Foley's catheters are tied over a bolster to give constant traction.
- ❑ Further anterior nasal packing and/or balloon inflation may be required to control nasal bleeding.
- ❑ Ongoing oral bleeding can be controlled by packing of oropharynx and oral cavity.
- ❑ The pack may be left for 48-72 hrs, if required. Prolonged pressure from pack may cause necrosis of soft palate.
- ❑ In some uncontrolled cases, bilateral external carotid artery ligation may be required.
- ❑ If facilities are available, angiographic embolization of maxillary artery branches can stop bleeding.
- ❑ Definitive maxillofacial surgery is undertaken after control of acute hemorrhage.

B. Breathing and Ventilatory Support

Once a clear airway has been obtained, the patient's breathing is assessed. The chest is exposed and rate and depth of respiration are measured. A respiratory rate of <10 or >30/min indicates a severe underlying problem. There are five life-threatening chest injuries that must be identified and treated during primary survey.

- i. *Tension pneumothorax*: Air enters the pleural cavity from bronchial injury. But air is unable to go back due to a valve mechanism leading to rapidly increasing pneumothorax. It can kill the patient within minutes. Clinical features are:
 - Respiratory distress "I can't breathe".
 - Hyperinflated chest (resonant on percussion).
 - Deviated trachea.
 - Decreased breath sounds.
 - Tachycardia.
 - Hypotension.

Needle thoracocentesis should be done immediately by putting 12G cannula in pleural cavity through 2nd intercostal space in midclavicular line. It should be followed by definitive chest tube placement that is connected to underwater seal drain.

- ii. *Massive hemothorax*: It is collection of more than 1500 ml blood into the pleural cavity. The patient

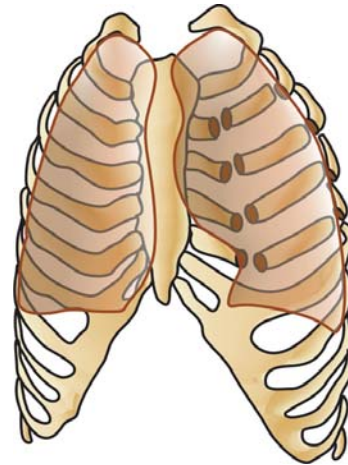


Fig. 10.5: Flail chest

may present in shock. Signs are similar to tension pneumothorax except for dullness on percussion. Treatment is intercostal tube drainage.

- iii. *Sucking chest wound*: It should be covered using dressing taped on three sides, allowing escape of air through a valve like action.
- iv. *Flail chest*: Two or more ribs are broken at two points leading to "paradoxical respiration". The flail segment moves in during inspiration and moves out during expiration (Fig. 10.5). There is underlying lung contusion and hypoxia. Patient may require tracheal intubation and positive pressure ventilation. There may be subcutaneous emphysema (surgical emphysema) due to lung injury requiring intercostal intubation (Fig. 10.6).



Fig. 10.6: Massive surgical emphysema following flail chest

- v. *Pericardial tamponade*: There is collection of blood in the pericardial cavity due to injury to the underlying heart. The patient usually does not reach hospital alive. The clinical signs are: Hypotension, muffled heart sounds and distended neck veins (**Beck's triad**). Needle pericardiocentesis should be performed and urgent thoracotomy arranged.

C. Circulation and Hemorrhage Control

- The best early signs of shock are pallor, cool clammy skin, tachycardia, anxiety and tachypnea.
- Hypotension is a late sign when >30% of blood volume is lost.
- Control external bleeding with direct pressure.
- Splint limb fractures.
- Insert two large bore cannulae (16 G in adults) in veins of ante-cubital fossa or forearm.
- If venous access not obtained, do cut-down on long saphenous vein at the ankle or median basilic vein in the arm.
- In children up to 10 years of age, intraosseous needle infusion is preferable to central venous access.
- Take blood samples for grouping and cross matching.
- Restore blood volume by rapid infusion of Ringer lactate solution (2 liters).
- Blood replacement by group specific cross matched blood or O-negative blood.
- If shock persists despite resuscitation, exclude non-hypovolemic causes of shock, e.g. cardiogenic shock, neurogenic shock.
- If investigations (chest X-ray, abdominal ultrasound, X-ray pelvis) suggest uncontrolled internal bleeding, consider exploration.
- Do constant monitoring of the patient with cardiac monitor, pulse oximeter, urine output measurement.
- The most important errors contributing to avoidable deaths are—failure to control bleeding and delay in operation.
- Outlines of hemorrhage control are given in Box 10.8.

Box 10.8: Management of circulation

Assess:	Consciousness level, skin color, temperature, pulse, BP.
Access:	Two peripheral intravenous lines.
Arrest:	External hemorrhage.
Ask:	Does patient require emergency surgery? (Thoracotomy/ Laparotomy/ Fracture fixation).
Attach:	Cardiac monitor, pulse oximeter, urinary catheter, Ryle's tube (if indicated).

D. Disability

A rapid neurological assessment is done at the end of primary survey to determine:

- Level of consciousness (Box 10.9).
- Pupillary size and reaction to light.
- Limb movement.

Box 10.9: 'AVPU' method of assessing level of consciousness

A	Alert
V	Responds to vocal stimuli
P	Responds to painful stimuli
U	Unresponsive

In case of unilateral fixed dilated pupil and neurological deficit, consult with a neurosurgeon immediately.

E. Exposure

- Completely expose the patient, usually by cutting off the clothes, so that complete examination can be performed.
- Log roll and examine the back.
- After completing the examination, cover the patient to prevent hypothermia.

SECONDARY SURVEY

The secondary survey involves taking a more complete history (**AMPLE**, Box 10.10) and making a detailed examination of the patient from head to toe. It covers (in this order):

- Head and scalp/maxillofacial
- Cervical spine and neck
- Chest
- Abdomen and pelvis
- Back and perineum
- Extremities
- Neurological—GCS score, complete sensory and motor assessment of upper and lower limbs.

Box 10.10: Essential points in history (AMPLE)

A	Allergies
M	Medication
P	Past medical history
L	Last food intake
E	Events related to injury

Head and Scalp/Maxillofacial Examination

- Examine scalp for lacerations and suture the bleeding wounds.
- Examine entire scalp/head for contusion, boggiess and fractures.
- Put gloved finger in scalp laceration for assessing a depressed fracture.
- Examine back of head when patient is log rolled.
- Look for signs of fracture base of skull viz. otorrhea, rhinorrhea, Battle's sign and raccoon eyes (See Chapter 17: Head Injury).
- Examine eyes—visual acuity, fundus and foreign bodies under lids.
- Palpate all bony prominences for depressed fractures.
- Examine nose, mouth, teeth and mandible
- Gloved finger inside mouth to feel for maxillary fractures (Le Fort I, II, III).
- Check midface mobility/loss of teeth/mandibular occlusion/ mandibular fractures.

Neck and Cervical Spine Examination

- Patients with head injury/maxillofacial trauma should be assumed to have unstable cervical spine injury.
- Do not remove a cervical collar until cervical spine has been assessed clinically and radiologically.
- A lateral cervical spine X-ray should be obtained during primary survey along with chest and pelvic X-rays.

- Undo collar with in-line immobilization and examine neck for:
 - ☐ Subcutaneous emphysema
 - ☐ Tracheal deviation
 - ☐ Laryngeal fracture
 - ☐ Arterial bleeding
 - ☐ Expanding hematoma
 - ☐ Penetrating neck wound
 - ☐ Airway compromise
- Do not explore neck wounds that penetrate the platysma in the emergency department.
- Examine cervical spine looking for midline tenderness, steps and open wounds.
- Replace collar unless cervical spine is cleared clinically and radiologically.

Examination of Chest and Other Parts

It should be done by General Surgery Registrar and details are beyond the scope of this book.

TERTIARY SURVEY

It should be completed within 24 hours of admission. It is review of previous diagnostic tests (blood tests, X-rays) and a thorough examination of the patient to ensure that all injuries have been identified.

11

Chapter

Tumors

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- A tumor is an independent and uncontrolled growth of new cells that serves no useful function.
- A tumor can be benign or malignant. The differences among the two types are shown in Box 11.1A.
- In between benign and malignant tumors, an intermediate group of tumors is also known. The tumors in this group are locally invasive, but usually don't spread by lymphatic or vascular route, e.g. pleomorphic adenoma of salivary glands, basal cell carcinoma.

Box 11.1A: Differences between benign and malignant tumor

Benign tumor	Malignant tumor
Slow growing	Rapidly growing
Well-capsulated	No definite capsule
Does not invade adjoining structures	Invades adjoining structures
No distant spread	Distant spread through lymphatics and blood stream
Good prognosis	Poor prognosis

BENIGN TUMORS

Adenoma

It arises from secretory glands, e.g. thyroid, parathyroid, breast. If an adenoma contains large amount of fibrous tissue, it is called *fibroadenoma* (commonly seen in breast).

If an adenoma has multiple cystic spaces, it is called as *cystadenoma* (seen in parotid, thyroid, pancreas, ovaries).

If an adenoma arises from secretory glands of mucous membrane, it is likely to be pedunculated (rectal polyp).

Fibroma

It is a benign tumor arising from fibrous connective tissue. A pure fibroma is rare and is mostly combined with other mesodermal tissues, e.g.

Nerve sheath	-	Neurofibroma
Glandular tissue	-	Fibroadenoma
Fat	-	Fibrolipoma
Muscles	-	Fibromyoma

A fibroma can be soft or hard depending upon proportion of fibrous tissue with other cellular tissue.

Papilloma

It is a benign tumor arising from epithelial surface (skin or mucous membrane). It consists of a central core of connective tissue containing lymphatics and blood vessels that is covered with epithelium. The surface may be rough or made of finger like projections. Depending upon its location, the surface epithelium can be:

- Squamous cell (skin, tongue, lip, cheek)
- Columnar cell (small and large intestine)
- Transitional cell (urinary bladder)

A papilloma of skin is of two types:

Squamous Cell Papilloma

It has four varieties:

- Congenital papilloma*: It is present since birth and is seen as brownish warty growth.
- Soft papilloma*: It is often seen on eyelids, neck and face of elderly people. It forms pedunculated, soft, fleshy skin tags.
- Keratin horn*: It is also seen in old people and is due to excess keratin formation.
- Infective papilloma*: It is due to viral infection caused by *Verruca vulgaris*. It is common in children and

young adults. It is usually seen at sites that are prone to trauma, e.g. beard area, hand, feet and genitals. It appears as small, pigmented nodules that unite to form frond-like surface. Most of the warts may disappear spontaneously.

Basal Cell Papilloma (Senile warts, Seborrheic keratosis)

It is a benign tumor due to overgrowth of basal layer of epidermis. They appear as raised, brownish warts over face, neck and shoulders in elderly people. They gradually increase in size but not in thickness. They may fall off spontaneously.

Treatment

Papillomas usually need surgical excision due to cosmetic reasons.

Lipoma

It is the most common benign tumor arising from fat cells of adult type (Box 11.1B).

It can occur anywhere in the body where fat is present, hence named universal tumor.

The most common sites are nape of neck, abdominal wall and thighs.

There are three types of lipoma:

Encapsulated Lipoma

Commonest variety present in subcutaneous tissue. The patient presents with painless slow growing swelling of long duration (Fig. 11.1).

- On examination, there is a soft smooth and lobulated swelling.

Box 11.1B: Lipoma

- Benign tumor arising from fat cells
- Universal tumor (except cranial cavity)
- Commonest site—nape of neck, abdominal wall
- Soft, smooth, lobular swelling
- Slipping sign present
- Pseudofluctuation present
- Pseudotransillumination present
- Treatment—surgical excision
- Lipoma in thigh or retroperitoneum may undergo malignant change



Fig. 11.1: Encapsulated lipoma at nape of the neck

- *Slipping sign:* If edge of the swelling is pressed, it slips under the finger. It is pathognomonic sign of lipoma and differentiates it from a cyst.
- *Pseudo-fluctuation:* A sense of fluctuation may be obtained since fat at body temperature behaves like fluid.
- *Pseudo-transillumination:* The swelling may transilluminate due to presence of clear fat.
- The swelling is free from overlying skin and freely mobile over underlying structures.
- Sometimes the swelling may become pedunculated.

Diffuse Lipoma (Fig. 11.2)

It is a rare variety and does not have characteristic features of lipoma hence called as 'pseudolipoma'. It is overgrowth of fat and does not have a capsule. It usually presents as a diffuse swelling at nape of the neck.

Multiple Lipomas

Sometimes subcutaneous lipomas are multiple and painful due to presence of nerve tissue (neurolipomas).



Fig. 11.2: Diffuse lipoma at back of thigh

The condition is known as *adiposis dolorosa* or *Dercum's disease*.

Lipomas are also classified according to their anatomical plane:

- i. *Subcutaneous*: Commonest variety with characteristic features described above.
- ii. *Subfascial*: It is difficult to diagnose because overlying fascia masks the lobulations and negates the slipping sign. Long standing subfascial lipoma deep to epicranial aponeurosis can erode the underlying bone.
- iii. *Intermuscular*: Commonly seen in thigh and becomes fixed on muscle contraction.
- iv. *Submucous*: Occurring under mucous membrane, e.g. in the tongue (causes macroglossia), in the larynx (causes respiratory obstruction).
- v. *Subserous*: Occurring beneath pleura or in retroperitoneum. It may attain enormous size without causing any symptoms due to presence of potential space.
- vi. *Extradural*: It is a rare spinal tumor presenting with cord compression. Intracranial lipomas do not occur due to absence of fat in the cranial cavity.
- vii. *Intraglandular*: Lipoma occurring within the glands, e.g. breast, pancreas, beneath renal capsule.
- viii. *Lipomas in relation to bones and joints*, e.g.
 - ☐ Subperiosteal
 - ☐ Subsynovial
 - ☐ Intra-articular

Complications

Long standing lipomas may undergo:

- Myxomatous degeneration
- Saponification
- Calcification

Large sized lipoma in thigh or retroperitoneum may undergo malignant transformation into liposarcoma. The swelling starts growing rapidly in size and becomes painful. Overlying veins become dilated and surface becomes warm due to increased vascularity. If untreated, overlying skin may ulcerate and fungate due to rapidly growing tumor.

Treatment

Lipoma is treated by surgical excision. Aim of surgery is to take care of cosmetic disfigurement and to prevent complications.

Neuroma

Benign tumor of nerve is called neuroma. Based on site of origin, they are of two types:

True Neuromas

They are very rare and arise from sympathetic nervous system. They develop from neural crest and their distribution is shown in Box 11.2.

False Neuromas

They are benign tumors arising from nerve sheath. This group includes:

- i. Neurilemmoma
- ii. Stump neuroma
- iii. Neurofibroma

i. *Neurilemmoma (Schwannoma)*: It is a benign tumor arising from Schwann cells. Commonest site of involvement is acoustic nerve. It produces a soft, whitish, lobulated mass that displaces the nerve from which it arises. It can be safely removed without damaging the nerve of origin.

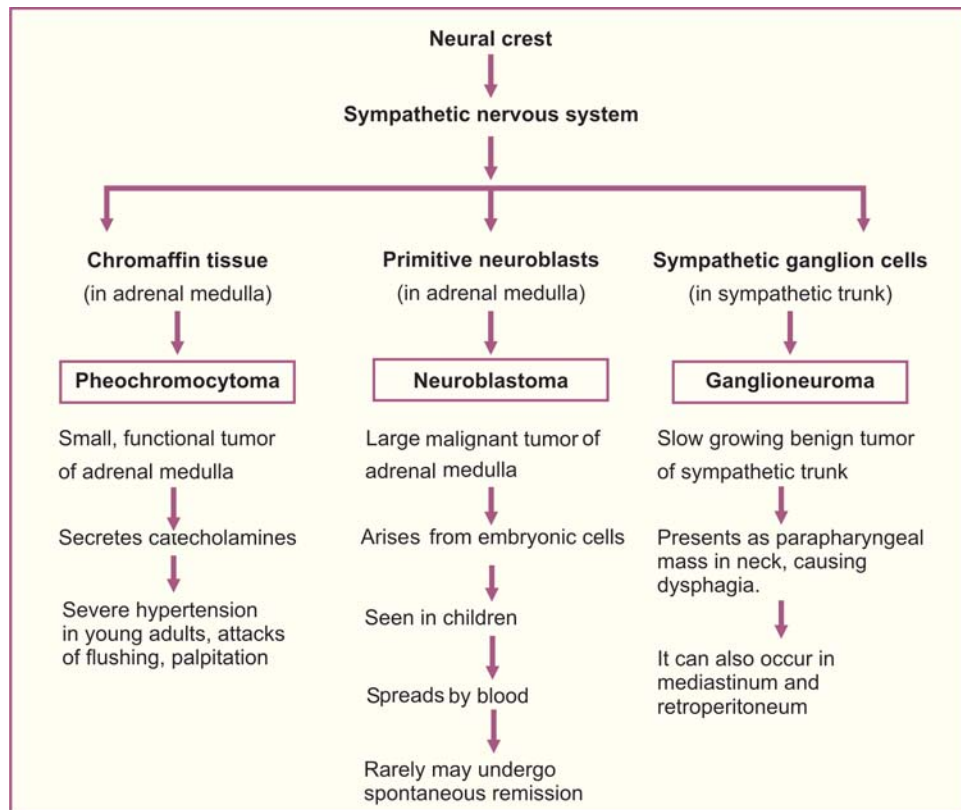
ii. *Stump neuroma*: After limb amputation, the end of a divided nerve forms a fusiform swelling due to proliferation of nerve fibers. It is also called as 'Amputation neuroma'. It can cause numbness, tingling and severe neuralgic pain due to pressure by prosthesis or nerve entrapment in the scar. Treatment is excision of neuroma. However, it can be prevented if nerve is divided at a higher level than the site of amputation. Other causes of neuralgic pain due to nerve compression are shown in Box 11.3.

iii. *Neurofibroma*: It arises from the connective tissue of nerve sheath (endoneurium). It can appear at any age but usually presents in adult life. As nerve fibers pass through the tumor, so tumor cannot be removed without damaging the nerve (c/f neurilemmoma). It has following types:

Localized Neurofibroma

- It is usually seen in subcutaneous tissue.
- It mostly involves peripheral nerves (ulnar or median nerve) or cranial nerves (acoustic neuroma).
- It produces a fusiform swelling in direction of nerve.
- Mostly asymptomatic, but patient may complain of paresthesia, numbness and pain in distribution of the nerve.

Box 11.2: True neuromas



Box 11.3: Causes of nerve compression

- Stump neuroma
- Cervical rib syndrome
- Carpel tunnel syndrome
- Elbow tunnel syndrome
- Tarsal tunnel syndrome
- Morton's metatarsalgia

Box 11.4: Complications of neurofibroma

- Sensory and motor weakness
- Deafness (Acoustic neuroma)
- Paraplegia (Dumb-bell tumor pressing spinal cord)
- Cystic degeneration
- Sarcomatous change

- On examination, there is 'tender subcutaneous nodule' that is firm, smooth and circumscribed.
- The swelling typically moves at right angle to the direction of nerve and fixed in the direction of nerve.
- The area of distribution of affected nerve should be examined for sensory and motor weakness.
- *Complications:* Box 11.4.
- *Differential diagnosis:* Box 11.5A.
- *Treatment:* Asymptomatic neurofibroma should be left as such since excision will always lead to the damage of involved nerve. Indications of excision are:
 - ☐ Cosmetic deformity.
 - ☐ Symptomatic; causing severe pain and paraesthesia.
 - ☐ Rapidly growing (? Sarcomatous change).

Box 11.5A: Differential diagnosis of neurofibroma

- Lymph node (in neck, multiple)
- Dermoid cyst (in midline)
- Sebaceous cyst (skin adherent)
- Lipoma (slipping sign)
- Hemangioma (skin discoloration, compressible)
- Ganglion (see Box 11.5B)

The swelling should be completely excised to prevent the risk of recurrence.

Generalized Neurofibromatosis (von Recklinghausen's disease)

- Multiple neurofibromas are seen involving various parts of the body (face, neck, trunk and limbs) (Fig. 11.3).

Box 11.5B: Ganglion

- Cause—myxomatous degeneration of tendon sheath or synovial lining of joint space.
- Site—commonly on wrist (dorsum of hand).
- Presentation—painless, smooth, tense cystic swelling containing gelatinous fluid.
- Mobile side to side (at right angle to tendon)
- Mobility gets restricted on contraction of the tendon
- Treatment
 - ☐ No treatment for asymptomatic ganglion.
 - ☐ Spontaneous rupture may cure it.
 - ☐ Needle aspiration and intra-lesional injection of hyalase and kanacort (steroid).
 - ☐ Surgical excision should be avoided due to risk of recurrence.

**Fig. 11.3: Generalized neurofibromatosis**

- It is an autosomal dominant disease and runs in the families.
- It may involve peripheral, spinal and cranial nerves.
- There may be associated pigmentation of skin called **Cafe-au-lait spots**. It is so named because its appearance resembles color of coffee diluted with milk (Fig. 11.4).
- One or more neurofibromas may undergo sarcomatous change.
- *Treatment:* Treatment is 'wait and watch' policy since excision of so many swellings is not possible. Sometimes, one or more swellings may need excision if there is:
 - ☐ neurological deficit (e.g. pressure on spinal cord).
 - ☐ Severe pain.
 - ☐ Suspicion of malignant change.
- von Recklinghausen's disease of bone is a separate entity (Box 11.6).

**Fig. 11.4: Cafe-au-lait spots****Box 11.6: von Recklinghausen's disease**

- A. *Generalized neurofibromatosis*
 - ☐ Multiple neurofibromas
 - ☐ Cafe-au-lait spots
- B. *von Recklinghausen's disease of bone*
 - ☐ Osteitis fibrosa cystica
 - ☐ Parathyroid adenoma causing hyperparathyroidism
 - ☐ Pathological fractures and renal stones

Plexiform Neurofibromatosis

- There is 'myxofibromatous degeneration' of endoneurium so that affected nerve becomes enormously thickened (Fig. 11.5).
- It usually involves branches of 5th cranial nerve (Trigeminal nerve) in area of face and scalp.
- The affected skin of face becomes thick, edematous, pigmented and adherent causing severe cosmetic deformity.

**Fig. 11.5: Plexiform neurofibromatosis**

- As it grows in size, the involved skin starts hanging down in pendulous folds (Pachydermatocele).
- The hanging skin folds can obstruct the vision.
- As a rule, the skin is covered with hair unless repeated friction causes skin ulceration and scarring.
- Treatment is staged excision. However, it is difficult and should be performed by a plastic surgeon.

Elephantiasis Neuromatosa

- It is advanced and severe form of plexiform neurofibromatosis.
- It usually affects lower limbs.
- The skin is coarse, dry and thickened. The subcutaneous tissue is also greatly thickened and fat is replaced by fibrous tissue.
- The appearance resembles elephant's hide and the patient finds walking very difficult.
- *Differential diagnosis:* Filarial elephantiasis (involves lymphatics).

Hemangioma: See Chapter 12—Cysts and Neck Swellings.

Lymphangioma: See Chapter 12—Cysts and Neck Swellings.

Hamartoma: See Chapter 12—Cysts and Neck Swellings.

Benign melanoma (Pigmented nevus):

- These are benign tumors arising from melanocytes (Figs 11.6 and 11.7).
- Melanocytes are derived from neural crest and are present in basal layer of epidermis.
- Proliferation of melanocytes produces pigmented nevus. It has following types:
 - i. *Lentigo:* It is present only in basal layer of epidermis.
 - ii. *Junctional nevus:* It is localized aggregation of melanocytes projecting into dermis. It appears as a brownish black, localized and slightly raised lesion anywhere on the body. It can undergo malignant change.
 - iii. *Dermal nevus:* It is present entirely in the dermis. It is mostly seen on face as 'hairy mole'. It is a pigmented, dome shaped, soft, smooth lesion. It never undergoes malignant change.
 - iv. *Compound nevus:* It is combination of both junctional and dermal nevus. It is usually seen in adults as a pigmented, rounded and elevated



Fig. 11.6: Pigmented nevus infra-orbital region



Fig. 11.7: Pigmented nevus forehead

lesion. Its junctional component is prone to malignant change.

- v. *Blue nevus:* It is seen on face, dorsum of hand and foot in babies. It is darkly pigmented and due to presence of overlying thin normal epidermis, it looks shiny and blue in color. Very rarely, it may undergo malignant change.
- vi. *Congenital nevus:*
 - A. *Hairy mole:* Common variety and does not change to malignancy.
 - B. *Giant lesion:* It may cover 25% or more of body surface area as irregular pigmentation. It can undergo malignant change (Fig. 11.8).
- vii. *Hutchinson's melanotic freckles:* These are seen as dark pigmented, smooth and flat lesions in elderly persons on sun exposed areas (face and neck). These have high incidence of malignant change (Fig. 11.9).



Fig. 11.8: Giant congenital nevus involving upper trunk and forehead



Fig. 11.9: Hutchinson's melanotic freckles on forehead

Treatment of Pigmented Nevus

Surgical excision. Indications are:

- Cosmetic reasons.
- Nevus exposed to repeated trauma, e.g. cuts during shaving, rubbing by clothes (belts, brassier, etc.).
- Suspicion of malignant change.

The excision should involve 2 mm of healthy skin margin and the specimen should always be sent for histopathological examination to rule out malignancy.



Fig. 11.10: Rhinophyma

Other Benign Tumors in Head and Neck Region

Turban Tumor (Cylindroma)

- It arises from apocrine glands and is benign in nature.
- It forms a slow growing extensive swelling that covers the scalp and looks like a turban.
- Surface ulceration is uncommon.
- Treatment is cryotherapy.

Potato Nose (Rhinophyma)

The skin of distal nose becomes thickened and bluish red in color. The openings of sebaceous follicles become prominent. Rarely, it may be associated with basal cell carcinoma. Treatment is surgical excision of excess tissue (Fig. 11.10).

Keratoacanthoma (Molluscum Sebaceum)

- It is a benign tumor arising from hair follicle.
- It is mostly seen on face and neck in young adults.
- It forms a small, solitary, hard tumor in subcutaneous tissue, attached to overlying skin.
- Clinically, it resembles sebaceous cyst.
- There is central dark brown area that separates spontaneously in 2-4 weeks time and it heals with scarring.

MALIGNANT TUMORS

- They are classified based on the cells of their origin (Box 11.7).

- A benign tumor may undergo malignant transformation. The clinical features suggesting malignant change in a benign tumor are shown in Box 11.8.
- Histopathological features, that help in differentiating benign and malignant tumors, are shown in Box 11.9.

Box 11.7: Classification of malignant tumors based on cell of origin

Origin	Type of malignancy
Epithelial	Squamous cell carcinoma, Basal cell carcinoma
Endothelial	Adenocarcinoma
Mesoderm	Sarcoma
Germ cells	Seminoma, Teratoma
Melanocytes	Malignant melanoma

Box 11.8: Signs of malignant change in a benign tumor

- Rapid increase in tumor size.
- Overlying skin becomes stretched with dilated cutaneous veins.
- Tumor becomes fixed to surrounding structures.
- Pressure effect/invasion of adjoining structures:
 - Facial palsy in pleomorphic adenoma (facial nerve involvement).
 - Hoarseness of voice in thyroid adenoma (Recurrent laryngeal nerve involvement).
- Metastasis:
 - To regional lymph nodes.
 - To distant organs by blood (liver, lungs, brain).
- Malignant cachexia:
 - Loss of weight and appetite

Box 11.9: Histopathological features of tumors

Benign

- Hypertrophy: Increase in cell size
- Hyperplasia: Increase in cell number

Malignant

- Metaplasia: Change in cell character, e.g. columnar epithelium changes to squamous epithelium
- Dysplasia: Change in intracellular characters, e.g. size and shape of cell as well as nucleus
- Carcinoma *in situ*: Intracellular characters resemble cancer but without invasion into extracellular matrix
- Anaplasia: Normally tumor cells resemble with the tissue of their origin. If there is complete loss of differentiation of cells, it is anaplasia and indicates aggressive cancer

- The *diagnosis* of malignancy is confirmed by pathological examination of the tissue that needs to be biopsied. Various methods of taking tissue biopsy are:
 - FNAB (Fine needle aspiration biopsy)*: It is minimally invasive and quickest procedure for making diagnosis of cancer. 23G needle is inserted into the tumor. Multiple passes are made with the needle through the tumor while maintaining suction with attached syringe. It breaks the tumor cells from the tissue and these cells are sucked into the needle. The cells are spread on a slide and examined under microscope after staining.
 - Incision/wedge biopsy*: When tumor is very large and appears to be inoperable, a wedge of tissue is taken from the margin of tumor including adjoining healthy tissue as well. The biopsy is not taken from the center of a large tumor since the area is likely to contain necrotic tissue.
 - Excision biopsy*: Small tumors are removed *in toto* and subjected to histopathology. This procedure is both diagnostic as well as therapeutic.
 - Core needle biopsy*: A core of tumor tissue is taken for biopsy using tru-cut needle.
 - Endoscopic biopsy*: Tumor is seen through endoscope and multiple biopsies are taken from its margin using a special forceps that passes through biopsy channel of the endoscope.

Etiology

Although exact etiology of malignant tumors is not known but certain etiological factors are known for causation of malignant tumors. These are:

- Genetic factors
- Environmental factors

Genetic Factors

The 'cell cycle' is under control of genes and if these controlling genes become diseased (mutated), it causes cancer (Box 11.10).

Box 11.10: Genes controlling various stages of cell cycle

Stage of cell cycle	Controlling genes
Proliferation of cells	Oncogenes (k-ras, c-myc)
Programmed cell death (apoptosis)	Tumor suppressor gene (APC, p53)

Various inherited malignancies due to genetic factors are:

- Familial breast cancer
- Familial ovarian cancer
- Familial melanoma
- Medullary carcinoma thyroid

Environmental Factors

These are known as 'carcinogens'.

- **Smoking:** Lung, upper aerodigestive system and urinary bladder cancer.
- **Alcohol:** Liver cancer.
- **Sun exposure (UV rays):** Skin cancer.
- **Radiation exposure:** Leukemia, thyroid cancer.
- **Diet:** Smoked, spicy food causing carcinoma esophagus.
- **Infections:** *H. pylori* causing stomach cancer, EB virus causing Burkitt's lymphoma, HIV causing Kaposi's sarcoma.
- **Chemicals:** Nitrosamines causing lung cancer, aromatic amines causing bladder cancer.

There are certain factors which protect against cancer (Box 11.11).

Box 11.11: Protective factors against cancer

- Fresh fruits (antioxidants)
- Green vegetables (antioxidants)
- Regular exercise

Spread of Malignant Tumors

It can occur in following ways:

- Direct spread:** Malignant tumor can invade adjoining structures.
- Lymphatic spread:** It can occur by:
 - Invasion:** Adjoining lymphatics are invaded by the malignant cells and these cells spread to draining lymph nodes.
 - Embolization:** Malignant cells invading lymphatic vessels can embolize with lymphatic circulation to distant lymph nodes.
- Hematogenous spread:** Malignant cells can invade the draining veins and embolize to distant organs, e.g. liver, lungs, brain, bone marrow.
- Implantation:** Malignant cells may get deposited on adjoining surface that is in close contact, e.g. carcinoma of lower lip involving upper lip (kiss cancer).

Staging of Malignant Tumors

Aims of staging are:

- To assess the prognosis of disease, e.g. early stage carcinoma has better prognosis than late stage carcinoma.
- To plan the treatment, e.g. early carcinoma can have curative treatment while advanced carcinoma can only have palliative treatment.
- For comparison of results of treatment in various centers world over.

The most widely accepted system of staging is **TNM system** that has been adopted by UICC (Union Internationale Contre le Cancer). TNM stands for:

T- Tumor (extent of primary tumor).

N- Nodes (extent of involvement of regional lymph nodes).

M- Metastasis (presence or absence of distant metastasis).

T and N stage are defined differently for various tumors depending upon their anatomical location and lymphatic drainage of affected organ. However, M stage is called as M_0 (absence of metastasis) or M_1 (presence of metastasis). If it is not possible to detect metastasis it is called as M_x .

Tumor Grading

It is based on degree of pleomorphism seen on histopathological examination of tumor. The tumor can be:

- Well-differentiated
- Moderately differentiated
- Poorly differentiated

It is complementary to TNM staging in deciding the prognosis.

TYPES OF MALIGNANT TUMORS

Sarcoma

- It is mesodermal in origin.
- It is mostly seen during first and second decades.
- It usually grows rapidly and spreads by blood stream.
- It is generally painless and confused with benign conditions like hematoma. Diagnosis is often delayed.
- On *clinical examination* it appears as a fleshy mass (Sarx:Flesh). Dilated veins may be seen in overlying skin. The consistency is variable depending upon

amount of fibrous tissue and vascular tissue present in it. On palpation, tumor is warm and pulsatile due to high vascularity. Types of sarcoma are shown in Box 11.12A.

Box 11.12A: Types of sarcoma

Cell of origin	Sarcoma
Fat cells	Liposarcoma
Fibroblasts	Fibrosarcoma
Osteoblasts	Osteosarcoma
Chondroblasts	Chondrosarcoma
Striated muscles	Rhabdomyosarcoma (Fig. 11.11)
Smooth muscles	Leiomyosarcoma
Blood vessels	Hemangiosarcoma
Lymph vessels	Lymphangiosarcoma
Nerves	Neurofibrosarcoma



Fig. 11.11: Rhabdomyosarcoma of chest wall

- *Treatment* is wide excision with surrounding healthy tissue (2-3 cm) to prevent recurrence. Incomplete excision has high recurrence rate (Fig. 11.12A). Other treatment modalities are radiotherapy and chemotherapy.
- Salient features of sarcoma are summarized in Box 11.12B.
- Comparison between carcinoma and sarcoma is shown in Box 11.13.

Basal Cell Carcinoma (Rodent ulcer)

- It is most common malignant skin tumor (Box 11.14A).

Box 11.12B: Sarcoma—salient features

Age	Children 20-40 years Elderly	Rhabdomyosarcoma Kaposi's sarcoma, synovial sarcoma, osteosarcoma Angiosarcoma, fibrosarcoma
History		Painless, rapidly growing soft tissue tumor
Location	Head and neck Retroperitoneum Extremities	Angiosarcoma, osteosarcoma Liposarcoma Liposarcoma, osteosarcoma, synovial sarcoma
Inspection		Diffuse swelling, dilated veins in overlying skin
Palpation		Nontender, warm, variable consistency, may be pulsatile
Regional lymph nodes		Usually not enlarged. May be enlarged in rhabdomyosarcoma, malignant fibrous histiocytoma, synovial sarcoma
Systemic Examination (For metastasis):		Liver enlarged, non-tender Lung findings Bony tenderness
Investigations		FNAC/Wedge biopsy (<i>to confirm diagnosis</i>) MRI of affected part (<i>to assess the extent</i>) X-ray chest, USG abdomen (<i>metastatic work up</i>)
Treatment		Wide excision Compartment excision Amputation RT/CT



Fig. 11.12: Recurrent fibrosarcoma after incomplete excision

Box 11.13: Comparison between carcinoma and sarcoma

Carcinoma	Sarcoma
Origin: Ectodermal or Endodermal	Mesodermal
Age: Middle or elderly age	Young age
Occurrence: Very common tumor	Less common tumor
Progress: Slow growing tumor	Rapidly growing tumor
Metastasis: Lymphatic metastasis common and occurs early. Blood-borne metastasis occurs late	Blood-borne metastasis is common and occurs early. Lymphatic metastasis is rare.
Treatment: Surgery is main treatment. Mostly radio-sensitive	Surgery is main treatment. Mostly radioresistant.

Box 11.14A: Malignant skin tumors

- Low grade tumors
- Diagnosed early due to their location
- Good prognosis
- Distribution

70%	Basal cell carcinoma
20%	Squamous cell carcinoma
5%	Malignant melanoma

- It arises from basal cells of pilosebaceous adnexa and occurs only in skin.
- It cannot occur in the mucosal surface having squamous epithelium (tongue, lips) due to lack of pilosebaceous adnexa in these areas.

- It is a tumor of low grade malignancy. Mortality is extremely rare but cosmetic disfigurement is the main consideration with basal cell carcinoma.
- It commonly affects white skinned people of elderly age having high exposure to sunlight (in Australia, New Zealand).
- **Site:** It is mostly seen on face above an arbitrary line joining ear lobule to the angle of mouth (sun exposed area). The commonest site is inner canthus of eye.
- It is also called 'tear cancer' because it is commonly seen in region of the face where tears roll down.
- **Types:**
 - Nodular:** Solid, non-fluctuant swelling with central depression and pearly appearance.
 - Cystic:** Blue-gray, semitranslucent, cystic nodule with a network of fiery red blood vessels on the surface.
 - Ulcerative:** Non-healing ulcer is the commonest presentation. Temporary healing occurs with crusting. But the crust breaks down with a sero-hemorrhagic discharge leading to recurrent ulceration. On examination, the margins of ulcer are raised and rolled out (like a motor car tyre) with central ulceration covered with scab.
 - Field fire type:** It grows rapidly leading to destruction and disfigurement of facial skin. It has irregular spreading edge with central scarring (Fig. 11.13).
- **Spread:** The tumor is slow growing and locally invasive, hence called **Rodent ulcer**. It gradually



Fig. 11.13: Rapidly spreading basal cell carcinoma causing facial disfigurement

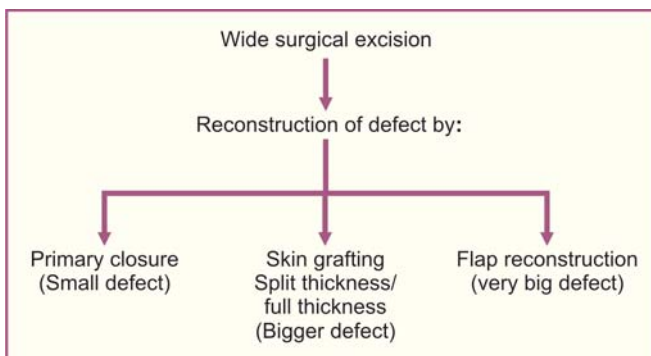
**Box 11.14B: Basal cell carcinoma—
differential diagnosis**

- Squamous cell carcinoma—everted margins
- Malignant melanoma—mimics pigmented basal cell carcinoma
- Keratoacanthoma—presents on face with ulceration and raised margins
- Sclerosing angioma

erodes deeper tissues like muscles, bone, cartilage, etc. and produces severe disfigurement.

Spread by lymphatics or bloodstream does not occur. Rarely basal cell carcinoma changes to squamous cell carcinoma in recurrent or neglected cases. In such situation, margins of the ulcer become everted and it spreads to regional lymph nodes.

- Differential diagnosis: See Box 11.14B.
- *Treatment: Surgery and Radiotherapy* are equally effective. The type of treatment is decided by the patient's condition and extent of disease.
 - In patients having extensive tumor eroding skull bones, radiotherapy is the treatment of choice. Dosage of radiotherapy is 4000-6000 rads.
 - In patients having localized lesion of the face, surgery is the treatment of choice (Box 11.14C). Tumor is excised with a healthy margin (3-5 mm). If the defect is small, it can be closed primarily. If the defect cannot be closed primarily, it should be covered with split or full thickness skin graft. On face, full thickness graft is taken from post-auricular skin and it gives better cosmetic result. Larger defect requires plastic reconstruction using pedicle flap.

Box 11.14C: Surgical management—basal cell carcinoma and squamous cell carcinoma

- *Moh's micrographic surgery*: This is a specialized dermatological technique meant to minimize tissue damage and to decrease disfigurement on areas like face. Visible tumor is excised in horizontal slices. The completeness of excision is confirmed by taking frozen sections from undersurface of excised lesion.
- *Cryosurgery and CO₂ laser* can be used for treating small lesions.
- Measures to prevent basal cell carcinoma include wearing protective clothing and sunscreen to prevent UV damage during sun exposure.

**Squamous Cell Carcinoma
(Epithelioma, Epidermoid Carcinoma)**

It arises from surfaces covered with squamous epithelium (skin, upper aerodigestive track, vagina). Sometimes surfaces not covered by squamous epithelium undergo a change to squamous type due to factors causing chronic irritation (squamous metaplasia), e.g.

- Transitional cell lining of urinary bladder undergoes squamous metaplasia by stones.
- Columnar cell lining of gallbladder undergoes squamous metaplasia due to gallstones.

In case of skin, squamous cell carcinoma arises from prickle cell layer of epidermis.

It is more common in skin of the face of elderly people.

It is more common in white skinned people.

It is more malignant and more rapidly growing than basal cell carcinoma. The differences between squamous and basal cell carcinoma are shown in Box 11.17C.

Premalignant skin lesions that can develop to squamous cell carcinoma are shown in Box 11.15.

Types

- Ulcerative—commonest presentation
- Proliferative—cauliflower like
- Ulceroproliferative

Clinical Features

- It commonly presents as non-healing ulcer that is progressively increasing in size.
- The ulcer is irregular in shape with everted and indurated edges. The base is indurated, attached to deeper structures and has a blood stained discharge (Figs 11.14 and 11.15).

Box 11.15: Premalignant skin lesions

- **Leukoplakia:** Small, circumscribed white plaque
- **Senile (solar) keratosis:** Prolonged sun exposure
- **Paget's disease**
- **Bowen's disease:** Well-defined brownish induration of skin
- **Radiodermatitis:** Exposure to X-rays
- **Lupus vulgaris:** Cutaneous tuberculosis
- **Chronic ulcers (Marjolin's ulcer)** (Box 11.16): Venous ulcer, keloid
- **Xeroderma pigmentosa**
- **Conditions causing chronic skin irritation:**
 - ❑ **Countryman's lip** is carcinoma lower lip in farmers due to sun exposure.
 - ❑ **Chimney sweep cancer** is carcinoma scrotum in chimney sweepers due to irritation by clothes soaked in oil or pitch.
 - ❑ **Kangri cancer** is carcinoma of abdominal wall due to Kangri (charcoal burner) applied by Kashmiris to abdominal wall for protection against cold.
 - ❑ **Kang cancer** is carcinoma of buttocks, heels and elbows due to sleeping on oven bed by Tibetans.

Box 11.16: Marjolin's ulcer

- Carcinoma developing in long standing ulcer/scar
 - Slow growing (because it is avascular)
 - Painless (because it has no nerves)
 - No metastasis to regional lymph nodes (because it has no lymphatics)
 - If it invades adjoining skin, it starts behaving as squamous cell carcinoma
 - Treatment: Wide excision.
 - It is radioresistant (because of avascularity)
- The diagnosis is confirmed by wedge biopsy from the margin of the ulcer that shows 'epithelial pearls' or 'cell nests'.

Spread

- Local spread to adjoining structures.
- Lymphatic spread to regional lymph nodes.
- Blood spread occurs only in very advanced stage.

Differential diagnosis: See Box 11.17A.



Fig. 11.14: Fungating, cauliflower like growth in the neck—squamous cell carcinoma



Fig. 11.15: Fungating growth involving sole—squamous cell carcinoma

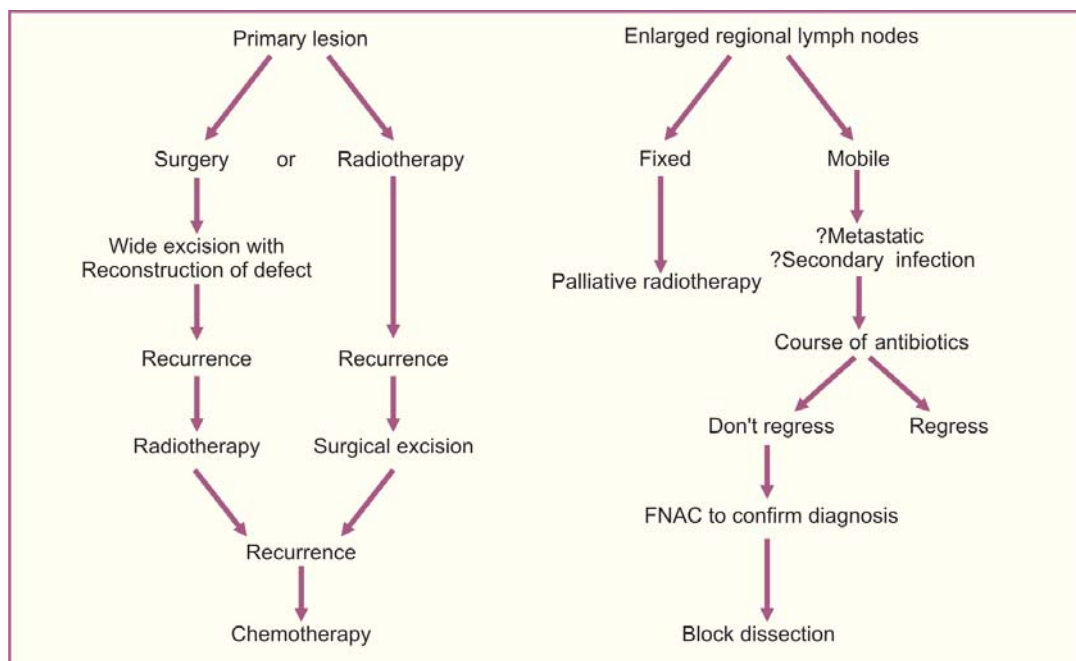
Box 11.17A: Squamous cell carcinoma—differential diagnosis

- Basal cell carcinoma
- Tubercular ulcer
- Syphilitic ulcer
- Chronic nonspecific ulcer
- Granuloma pyogenicum
- Keratoacanthoma

Treatment (Box 11.17B)**Treatment of primary lesion:**

- Surgery and radiotherapy are equally effective.
- Principles of local treatment are same as for basal cell carcinoma (see Box 11.14C).

Box 11.17B: Outlines of treatment—squamous cell carcinoma



Box 11.17C: Comparison between squamous and basal cell carcinoma

	<i>Squamous cell carcinoma</i>	<i>Basal cell carcinoma</i>
Incidence	Less common than basal cell carcinoma	Commonest skin malignancy
Origin	Prickle cell layer of epidermis	Basal cell layer of epidermis
Etiology	Chronic irritation	UV rays
Site	Any part of skin. Internal organs like gallbladder, urinary bladder (due to squamous metaplasia)	Mostly on sun exposed area of face
Tumor grade	High grade tumor, grows rapidly	Low grade tumor, grows slowly
Clinical findings	Irregular ulcer with everted edges	Rounded ulcer with raised and rolled out edges.
Histopathological findings	Solid columns of epithelial cells growing down into the dermis. Presence of 'epithelial pearls' or 'cell nests'.	Basaloid appearance of epithelial islands.
Spread	Locally invasive, spread to regional lymph nodes common.	Locally invasive, does not spread by lymphatic or hematogenous route.
Treatment	Surgery and RT. Involved lymph nodes need block dissection.	Surgery/RT. Regional lymph nodes do not need any treatment since they are not involved

- Tumor should be excised with healthy margin of 1-2 cm.
- Chemotherapy is also useful in advanced cases.

Treatment of metastatic lymph nodes:

- The regional lymph node enlargement can be due to secondary infection of the ulcerated growth. In

such case, the decision for surgery should be taken only if lymph nodes do not regress with antibiotic treatment.

- Diagnosis of metastatic deposits in lymph nodes is confirmed by FNAB.
- The treatment is block dissection of regional lymph nodes.
- If lymph nodes are large and fixed, palliative RT should be given.

Malignant Melanoma

It is a malignant tumor arising from melanocytes.

Malignant melanoma of skin is regarded as carcinoma (Melanocarcinoma). It has very high rate of metastasis.

Origin: It may arise *de novo* in normal skin or malignant change may occur in a pre-existing mole (Box 11.18).

Predisposing factors: Shown in Box 11.19.

Box 11.18: Features of malignant change in pre-existing mole

- Increase in size of mole
- Pigmentation becomes deep
- A halo of pigmentation appears in surrounding skin
- Itching
- Ulceration
- Bleeding
- Scab formation
- Enlargement of draining lymph nodes

Box 11.19: Predisposing factors for malignant melanoma

- UV rays
- White race
- Age: After puberty
- Sex: More in females
- Genetic predisposition
- Trauma
- Pre-existing mole

Site

Common sites: '**BANS**' area (Back, Arms, Neck and Scalp), lower legs in females.

Rare sites: Eyes, meninges, anal canal.



Fig. 11.16: Superficial spreading melanoma involving sole

Classification

i. Lentigo maligna melanoma:

- It is malignant change occurring in Hutchinson's melanotic freckles.
- Malignant change occurs in 10 years or more.
- It is seen in 6-8th decade.
- Relatively less aggressive.
- It appears on parts exposed to sun.

ii. Superficial spreading melanoma:

- Most common but less aggressive lesion.
- It can occur anywhere but more common on exposed parts of the body.
- Intradermal spread of tumor occurs in radial (Horizontal) direction.
- It presents as a flat, irregular pigmentation of skin (Fig.11.16).

iii. Nodular melanoma:

- Less common but more malignant.
- It can occur anywhere but mostly seen in genital and anal region.
- It mainly grows vertically and there is little radial growth.
- It presents as small, circumscribed, pigmented nodule that itches, ulcerates and bleeds (Fig. 11.17).
- Metastasis occurs early.

iv. *Acral lentiginous melanoma:* It is commonly seen on palm, sole and under the nail (subungual melanoma). Subungual melanoma begins as an area of pigmentation in the nailbed. The



Fig. 11.17: Nodular melanoma neck

pigmentation increases and raises the nailbed. The tumor may extrude through nail plate leading to ulceration and bleeding.

- v. *Amelanotic melanoma*: Usually malignant melanoma is a pigmented lesion but sometimes it has no or very little pigment, hence called amelanotic melanoma. It carries poor prognosis due to delay in the diagnosis. The cases usually present with regional lymph node metastasis.

CASE SUMMARY

50 years male presented with painless enlargement of left groin nodes for one year. There was no response to antibiotics and FNAC done twice was inconclusive. On careful examination of left lower limb, a painless, pigmented lesion was seen involving left sole that turned out to be malignant melanoma (Fig. 11.16). The enlarged groin lymph nodes were due to metastatic deposits. The patient underwent amputation foot with block dissection of groin nodes.

Learning Point: In case of lymphadenopathy, always examine the drainage area carefully.

Staging

Staging is done for planning treatment and to assess prognosis. Various methods of staging are:

1. *Clinical staging*: It is the simplest method.

- Stage-I Primary tumor only
- Stage-II Enlargement of regional lymph nodes
- Stage-III Distant metastasis to lungs, liver, brain, bones, etc.

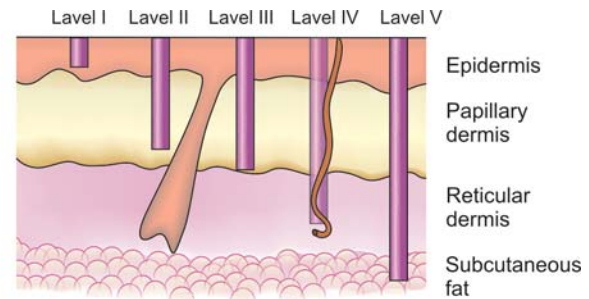


Fig. 11.18: Clark's level of tumor invasion

2. *Clark's level of tumor invasion*: This staging is done after histopathological examination of excised tumor specimen (Fig. 11.18).

- Level-I Tumor cells in epidermis above basement membrane.
- Level-II Tumor invading papillary dermis.
- Level-III Tumor at junction of papillary and reticular dermis.
- Level-IV Tumor invading reticular dermis.
- Level-V Tumor invading subcutaneous fat.

3. *Breslow's thickness of lesion*: Staging is done by measuring the maximum vertical thickness of melanoma at its center using optical micrometer.

- Stage-I Thickness 0.75 mm or less
- Stage-II 0.76 to 1.5 mm
- Stage-III 1.51 to 3.0 mm
- Stage-IV More than 3.0 mm

Breslow's tumor thickness is considered to be more practical and reliable indicator of prognosis than level of invasion. However, it is difficult to apply in ulcerated lesion. Its comparison with Clark's level of invasion is shown in Box 11.20.

Box 11.20: Comparison of classification methods

Clark's level of invasion	Breslow's tumor thickness	Prognosis
Level-I	0.75 mm	Low-risk group. Do not metastasize
Level II	0.76-1.5 mm	Intermediate risk group Metastasis in 25% cases
Level-III		
Level-IV	>1.5 mm	High-risk group. Metastasis in 60% cases
Level-V		

Spread

- i. *Local extension.*
- ii. *Lymphatic spread:* It occurs to regional lymph nodes by embolization. In case of lymphatic spread by permeation, 'satellite nodules' appear between primary tumor and regional lymph nodes.
- iii. *Hematogenous spread:* It occurs to liver, lungs, brain, bones and skin.

Prognostic factors: Shown in Box 11.21.

Box 11.21: Prognostic factors

- Poor prognosis seen in:
 - Male sex
 - Black race
 - Primary lesion of head and neck
 - Advanced clinical stage
 - Ulceration
 - Satellite nodules
 - Tumor thickness >1.5 mm
 - High level of invasion (level IV and V)

Clinical Examination

- Pigmented skin lesion, usually black in color.
- The size is variable and margins are usually irregular.
- Surface may be flat or raised above the skin (nodular).
- There may be ulceration in the center covered by crust.
- Consistency is firm.
- Mobility: The tumor arises from skin and can be lifted from deeper structures.
- Surrounding skin: There may be a pigmented halo around the primary lesion and 'satellite nodules' may be seen.
- Regional lymph nodes may be enlarged.
- There may be hepatomegaly (liver metastasis), pleural effusion (lung metastasis), neurological deficit (brain metastasis) and pathological fracture of long bones (bony metastasis).

Differential diagnosis: See Box 11.22.

Investigations

- Diagnosis is confirmed only by *excision biopsy*. Other investigations are done based on suspicion of metastasis.

Box 11.22: Differential diagnosis of malignant melanoma

- Pigmented basal cell carcinoma
- Cafe-au-lait spots
- Cavernous hemangioma
- Pigmented senile warts
- Blue nevus
- Kaposi's sarcoma
- Peutz-Jegher's syndrome: Familial condition. There is circumoral pigmentation with multiple intestinal polyps.

- *Lymphangiography* to look for lymph node metastasis. It is technically difficult and gives high false positive or negative results, hence not used commonly.
- *FNAC* of enlarged regional lymph nodes is very accurate in picking up metastasis. Open biopsy of lymph node should be avoided to prevent tumor spillage.
- *Ultrasound abdomen* for liver metastasis.
- *Chest X-ray* for pulmonary metastasis (canon ball shadow, pleural effusion).
- *CT/MRI head* for brain metastasis.
- *Bone scan* for bony metastasis.

Treatment

Treatment of Primary Lesion (Stage-I):

Surgical excision is the treatment of choice. There is no role of RT or CT as it is resistant to both. The surgical excision should include 1-2 cm of adjoining healthy skin. The depth of excision should not go beyond deep fascia since it limits the local recurrence. The excision should be elliptical in shape (along line of Langer) to allow tension free closure. The residual defect is closed primarily or with skin graft or with flap reconstruction.

Subungual melanoma is treated by amputation of the digit.

Melanoma of sole of the foot is treated by wide excision/amputation.

Treatment of Regional Lymph Nodes (Stage-II):

Clinically involved lymph nodes require block dissection. If lymph nodes are situated near the primary lesion, block dissection is done 'in continuity' with excision of primary tumor so as to remove 'in transit' deposits. If lymph nodes are far away from primary lesion, then two areas are removed through separate incisions. If lymph

nodes are fixed and inoperable, then palliative radiotherapy is given.

Treatment of Metastatic Malignant Melanoma (Stage-III):

Aim of treatment is palliation only.

- i. Radiotherapy for cerebral and bony metastasis
- ii. Chemotherapy: Drugs used are DTIC, vincristine, cisplatin.
- iii. Immunotherapy: Using BCG, Levamisol, Interferons, monoclonal antibodies.

Spontaneous regression is sometimes known to occur in malignant melanoma due to development of antibodies by body's natural defense mechanism.

Glandular Carcinoma

It arises from endoderm.

It arises from sites containing glandular tissue, e.g. breast, thyroid, alimentary tract, etc. Its types are:

- i. *Carcinoma simplex*: Cells are arranged in circumscribed groups and glandular structure is not identified, e.g. carcinoma breast.
- ii. *Adenocarcinoma*: Cells are arranged in form of acini and resemble with gland of their origin, e.g. intestinal adenocarcinoma.
- iii. *Colloid (muroid) carcinoma*: It arises from mucin secreting cells. The mucin permeates the stroma and gives gelatinous appearance, e.g. carcinoma colon.

Lymphoma

See Chapter 13: Diseases of Lymphatic System and Lymph Nodes.

12

Chapter

Cysts and Neck Swellings

Sham Singla, Sanjay Marwah

CYST

- The word 'cyst' is derived from a Greek word that means 'bladder'.
- It is defined as a swelling consisting of a sac filled with fluid and lined by epithelium, endothelium or granulation tissue.
- It can be a **true** or **false** cyst depending on the lining.
- A true cyst is lined by epithelium. A false cyst is a walled off collection (not lined by epithelium) generally lined by granulation tissue and is usually inflammatory or degenerative in origin, e.g. dental or radicular cyst, pancreatic pseudocyst.

Classification of Cysts (Box 12.1)

Box 12.1: Classification of cysts

Type	Mechanism	Example
Congenital cysts	Cysts of embryonic remnants	Thyroglossal, branchial, urachal
	Ectopia of various tissues	Dermoid, enterogenous
	Failed connection of tubular elements	Polycystic kidney
	Hamartomas	Cystic hygroma, hemangioma
Acquired cysts	Retention	Mucous cyst in oral cavity
	Implantation	Dermoid cyst
	Degeneration	Dental cyst
	Traumatic	Hematoma
	Hyperplastic	Fibrocystic disease of breast
	Neoplastic	Cystic teratoma
	Parasitic	Cysticercosis, hydatid cyst

Clinical features are same as that of a 'swelling'.

Symptoms

- Duration:** Congenital cysts are present since birth, but some may manifest later, e.g. branchial cyst.
- Mode of onset:** Spontaneous or following some disease or trauma (traumatic cyst—hematoma).
- Progress:** Slow growing or fast growing. Inflammatory cysts become very large in a few days time and may regress with antibiotics. However, neoplastic cysts are relatively slow growing and usually do not regress in size.
- Local effects:**
 - Pain due to nerve compression.
 - Dyspnea due to tracheal compression.
 - Dysphagia due to esophageal compression.
- Systemic effect:** Fever and toxemia in case of inflammatory cyst. Loss of weight and appetite, cachexia are features of malignant cyst.

Signs

Site: Dermoid cyst is in midline or at lines of embryonic fusion.

Size: Small or big. Exact dimensions need to be measured.

Shape: Cysts are usually round or hemispherical in shape.

Number: Sebaceous cysts are often single but multiple on scrotum.

Surface: Cysts are usually smooth surfaced.

Temperature: Local temperature is raised in an inflammatory lesion.

Tenderness: Inflammatory cysts are tender while neoplastic cysts and other cysts are nontender.

Consistency (Box 12.2): A cystic swelling is usually soft in consistency. In case fluid in the cyst is under tension, it feels firm. The cystic swelling is fluctuant due to presence of fluid in it.

Box 12.2: Description of various consistencies

Soft	Like feel of ear lobule.
Firm	Like feel of tip of the nose.
Hard	Like feel of olecranon process at elbow.
Cystic	Feels soft and fluctuant.
Tense cystic	Feels firm and smooth.

Fluctuation Test

Fluctuation means presence of transmitted impulse in two planes at right angles to each other. Presence of fluctuation in only one plane is fallacious, e.g. in quadriceps muscle in thigh, impulse can be elicited in transverse direction, but is absent in longitudinal axis of limb.

Steps of fluctuation test:

- A big movable lump needs to be fixed by middle finger and thumb of both the hands of examiner or an assistant before eliciting fluctuation.
- The pulp of the tip of right forefinger (watching finger) is placed halfway between center and periphery



Fig. 12.1A: Thumb and middle finger of two hands are fixing the swelling while pulps of two index fingers are eliciting the impulse

phery of swelling and is kept motionless throughout the procedure.

- The left forefinger (displacing finger) is placed upon a point at an equal distance from the center, diagonally opposite the right forefinger.
- On exerting pressure by 'displacing finger', the 'watching finger' feels transmission of impulse (Fig. 12.1A).
- The test is repeated in a plane right angle to the first plane.

Fallacies of fluctuation test:

- Lipoma** appears to fluctuate because fat is semi-fluid at body temperature. On careful clinical examination, one can appreciate that margin of a lipoma slips under the finger but it does not yield. However, margin of a cyst yields but does not slip under the finger.
- In a swelling less than 2 cm in size, fluctuation test is unreliable. *Paget's test* is helpful in such cases (Fig. 12.1C). A cystic swelling feels soft at the center and firm at the periphery. A solid swelling feels more firm at center than at periphery.

Transillumination Test

A few cysts are brilliantly translucent due to presence of clear fluid, e.g. cystic hygroma, ranula. Cysts containing thick pultaceous material are not translucent, e.g. dermoid cyst, sebaceous cyst.

Steps of transillumination test: The test should be performed in a darkroom using bright pinpoint light source (pencil torch). If test is performed in a well lit



Fig. 12.1B: A brilliantly transilluminant swelling

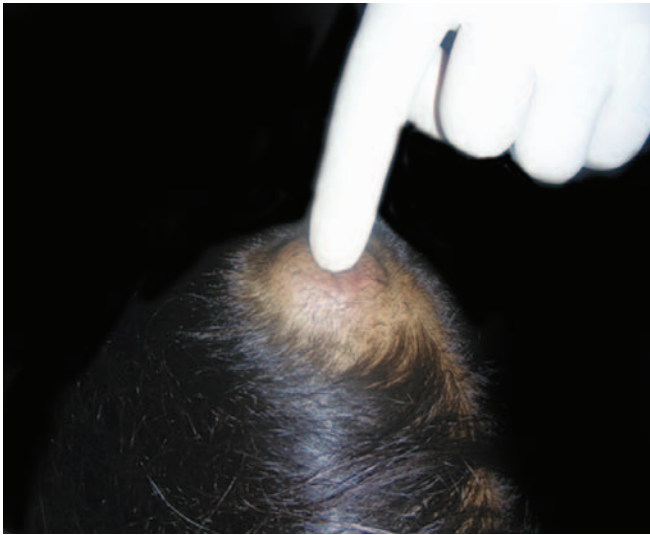


Fig. 12.1C: Paget's test for fluctuation in a small scalp swelling

room using a broad light source (flash light), it is bound to fail. The light should be placed on one side of the cyst and not directly on top of it (Fig. 12.1B). Since light travels in a straight line, so in a transilluminant swelling it can be seen in an area diagonally opposite to the point of contact with light source.

Mobility: The cysts in subcutaneous plane are usually mobile, e.g. dermoid cyst. However, sebaceous cyst has restricted mobility due to adherence with overlying skin especially at punctum. The cysts in muscle planes are mobile when muscle is lax and become immobile on muscle contraction, e.g. cysticercosis in masseter muscle, sternomastoid muscle. The cysts deep to the muscle have restricted mobility and become less prominent and immobile on muscle contraction, e.g. branchial cyst.

Steps to determine relation of a cystic swelling in neck to sternomastoid (Fig. 12.1D):

- Stand behind the patient.
- Ask him to turn his face in opposite direction (e.g. towards left in right sided swelling) against resistance of palm of your hand.
- Feel the anterior border of sternomastoid and appreciate its contraction and relation with the neck swelling.

Pulsations

Expansile impulse is felt in an aneurysm and *Transmitted impulse* is felt when swelling is overlying a vessel. Place



Fig. 12.1D: Determining relation of neck swelling to the sternomastoid muscle

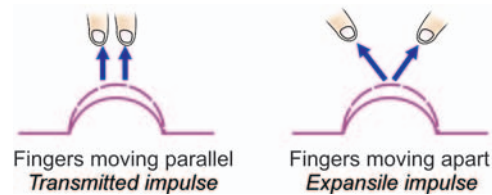


Fig. 12.2A: Difference in transmitted and expansile impulse

index and middle finger over the swelling. They will be felt to move with the swelling. If the pulsation is transmitted, the finger movements are parallel with each other. If the swelling is expansile the fingers are felt to move apart (Fig. 12.2A). The causes of pulsatile swelling in neck are given in Box 12.3A.

Box 12.3A: Pulsatile swelling in neck

- Carotid artery aneurysm (Expansile).
- Carotid body tumor (Transmitted pulsation).
- Lymph node mass over carotid artery (Transmitted pulsations).
- Subclavian artery aneurysm (Expansile).

Compressibility: On compression, the swelling diminishes in size considerably or even disappears (Fig. 12.2B). On releasing pressure, it refills slowly. It is characteristically seen in cavernous hemangioma due to communicating blood vessels. Causes of compressible swellings are given in Box 12.3B.

Bruit: It is heard as machinery murmur on auscultation in case of AV fistula.



Fig. 12.2 B: Testing for compressibility

Box 12.3B: Compressible swellings

- Cavernous hemangioma
- AV malformation
- Aneurysm
- Meningocele

Complications in a Cyst

1. **Infection:** The cyst may become inflamed due to superadded bacterial infection. If untreated, it may turn into an abscess and burst outside forming an ulcer or sinus, e.g. sebaceous cyst.
2. **Hemorrhage:** The cyst becomes painful and suddenly becomes very large in size, e.g. hemorrhage in a thyroglossal cyst. If not evacuated urgently, it may press on trachea and can cause respiratory obstruction.
3. **Torsion:** A large pedunculated cyst may undergo torsion on its axis, e.g. torsion of ovarian dermoid presenting as acute abdomen.
4. **Malignant transformation:** In teratomatous dermoid.
5. **Calcification:** In long standing cases, cyst wall may undergo calcification, e.g. hydatid cyst, cysticercosis, sebaceous cyst of scrotum.
6. Cystic swellings arising from various anatomical structures in the head and neck region are given in Box 12.4.

Box 12.4: Cystic swellings in head and neck region

Skin and subcutaneous tissues	Dermoid cyst, sebaceous cyst
Blood vessels	Hemangioma, aneurysm, hematoma
Lymphatics	Cystic hygroma
Lymph nodes	Cold abscess
Muscles	Cysticercosis
Thyroid gland	Cystic adenoma, Thyroglossal cyst
Branchial arch remnant	Branchial cyst
Pharynx	Pharyngeal pouch
Larynx	Laryngocele, subhyoid bursa
Salivary glands	Parotid abscess, Hamartomas, retention cyst (minor salivary glands)
Lacrimal sac	Lacrimal sac mucocele
Oral cavity	Ranula Mucous cysts
Odontogenic cysts	Dental cyst Dentigerous cyst Adamantinoma, Keratocyst
Non-odontogenic cysts–	Nasolabial cyst Nasopalatine cyst Median cyst Globulomaxillary cyst Solitary bone cyst
Meninges	Meningocele (occipital region, root of nose)

DERMOID CYST

Dermoid is a cyst lined by squamous epithelium. It contains pultaceous (tooth paste like) material that arises from degenerated and desquamated epithelial cells.

Types of Dermoid

i. Sequestration Dermoid

As name implies, it is formed by inclusion of surface epithelium at line of fusion of dermatomes. Common sites are:

- In midline of the body more so in head and neck region (sublingual dermoid) (Fig. 12.3).



Fig. 12.3: Huge sublingual dermoid pushing the tongue back



Fig. 12.5: Preauricular dermoid



Fig. 12.4: Internal angular dermoid



Fig. 12.6: Occipital dermoid

- External angular dermoid at outer canthus of the eye (lines of fusion of frontonasal and maxillary processes).
- Internal angular dermoid at root of the nose (Fig. 12.4).
- Pre-auricular and postauricular dermoid—in front and behind the ear respectively (site of fusion of auricular tubercles) (Fig. 12.5).
- On the head at sites of fusion of skull bones, e.g. occipital dermoid (Fig. 12.6).

Clinical features:

- Painless slow growing swelling presenting at young age (10-25 years).
- The surface is smooth (no punctum seen, cf. sebaceous cyst).
- The cyst is not attached to overlying skin (cf. sebaceous cyst).
- The cyst is often free from underlying structures.
- The cyst feels soft and may indent on pressure due to presence of pultaceous material.
- The cyst is non-transilluminant.
- In case of external or internal angular dermoid, the cyst may even erode the underlying bone and

become attached to dura mater. Sometime cyst may have a dumb-bell extension into the orbit or skull.

- X-ray skull may show a depression or gap in the underlying bone.
- If intracranial extension is suspected, CT scan should be done.
- Treatment is excision of cyst.
- If there is intracranial extension, excision should be done under GA by raising an osteoplastic flap.

Sublingual Dermoid

It is a type of sequestration dermoid formed by inclusion of surface epithelium at line of fusion of first branchial arches or mandibular arches.

- It can either be median or lateral, and either above or below the mylohyoid muscle.
- Median variety is more common than lateral variety.
- The cysts above mylohyoid present as a swelling in the floor of mouth below tongue and needs to be differentiated from ranula (see Fig. 12.3). The dermoid cyst is non-transilluminant while the ranula is brilliantly transilluminant (Box 12.5).
- The cyst below mylohyoid presents as a submental swelling (double chin appearance) and needs to be differentiated from thyroglossal cyst. The latter moves on deglutition as well as on protrusion of tongue.
- Treatment is surgical excision. The median dermoid is removed through submental incision. The lateral dermoid lying above mylohyoid is removed through floor of the mouth and the one lying below mylohyoid is removed through a submandibular incision.

Box 12.5: Sublingual dermoid

- *Above mylohyoid*
 - Non-transilluminant
 - D/D: Ranula (brilliantly transilluminant)
- *Below mylohyoid*
 - Does not move with tongue protrusion or deglutition
 - D/D: Suprahyoid thyroglossal cyst (moves with tongue protrusion and deglutition)

ii. Implantation Dermoid

- Following a puncture injury with a needle or thorn (usually in digits), a fragment of epidermis is driven



Fig. 12.7: Implantation dermoid ring finger



Fig. 12.8: Implantation dermoid ear lobule at puncture site of earring

beneath the dermis and continues to proliferate to form a cyst (Figs 12.7 and 12.8).

- It is commonly seen in farmers and tailors who are more prone to such injury.
- A small tense cystic swelling develops at the site of injury.
- Treatment is surgical excision.

iii. Teratomatous Dermoid

It arises from totipotent cells containing cells from all three embryonic layers, i.e. ectoderm, mesoderm and endoderm. So, it may contain elements arising from these germ layers like hair, teeth, bone, cartilage, muscle, glands and cheesy material.

- Common sites are testis, ovary, superior mediastinum, retroperitoneum and presacral area.
- These dermoids may undergo malignant change (carcinoma or sarcoma).

iv. *Tubulo-embryonic Dermoid*

- The cyst develops from unobliterated part of congenital ectodermal tube.
Examples are thyroglossal cyst, ependymal cyst of brain, post-anal dermoid.
Thyroglossal cyst: See Chapter 23: The Thyroid Gland.

SEBACEOUS CYST

- It is a cystic swelling in the skin occurring due to obstruction of a opening of sebaceous duct. Thus, it is a retention cyst.
- Pathologically, it is called as *epidermoid cyst* because it is lined by *superficial squamous cells*.
- Common sites are face, scalp, back and scrotum.
- It can occur anywhere except on the palms and soles where sebaceous glands are missing.
- Cysts are usually multiple in scalp and scrotum (Figs 12.9 and 12.10).



Fig. 12.9: Multiple sebaceous cysts scalp

Clinical Features

- Slow growing, small, painless swelling in the skin.
- It presents as a hemispherical swelling, nontender, firm in consistency with no definite edge.



Fig. 12.10: Multiple sebaceous cysts scrotum

- Due to small size and pultaceous contents, it is usually not possible to elicit fluctuation test.
- When swelling is indented with finger, it stays indented due to pultaceous contents.
- Presence of bluish spot or punctum (site of duct blockage) usually clinches the diagnosis.
- Sebaceous cyst is always fixed to the overlying skin (cf. dermoid cyst that is free from skin, Box 12.6).
- The cyst is free from underlying structures.
- The cyst is non-transilluminant.
- *Treatment* is total excision of the cyst.
- If cyst is infected, it should be treated with antibiotics first. Otherwise there is risk of incomplete removal.
- There are two ways to remove the cysts:
 - a. *Incision-avulsion method*: Under local anesthesia, an incision is made at most prominent part of the cyst including skin and cyst wall. The cyst is evacuated by squeezing its contents. The cyst wall is then held with an artery forceps and gradually avulsed.
 - b. *Dissection method*: An elliptical incision is made on the summit of cyst including the punctum. The skin flaps are raised and cyst is dissected intact and removed.

Complications

- a. *Infection*: It is the commonest complication. The cyst becomes large and painful showing signs of acute inflammation (Fig. 12.11). Treatment is antibiotics (amoxycloxacillin) and the cyst should be excised

Box 12.6: Sebaceous cyst vs dermoid cyst in head and neck region

	<i>Dermoid cyst</i>	<i>Sebaceous cyst</i>
Etiology	Congenital, inclusion of surface epithelial cells	Acquired, retention cyst due to blocked duct of sebaceous glands
Site	At lines of fusion of dermatomes, usually midline.	Anywhere except palms and soles
Skin punctum	Absent	Present in many cases, diagnostic
Overlying skin	Freely mobile	Fixed
Underlying structures	Freely mobile, underlying bone may be indented	Freely mobile, underlying bone is not indented.
Intracranial extension	May occur sometimes	Never occurs
Infection	Rarely occurs	Common complication
Treatment	Excision	Incision-avulsion or excision

**Fig. 12.11:** Infected sebaceous cyst**Fig. 12.12:** Sebaceous horn penis

once infection settles. If infection does not settle with antibiotics, treatment is incision and drainage of pus (like an abscess). Later incision and avulsion of cyst wall is done.

- b. **Ulceration:** An infected cyst may rupture to discharge its contents and an ulcerated surface is left. If a large sebaceous cyst of scalp ulcerates, excessive granulation tissue may form that looks like an epithelioma. It is called as *Cock's peculiar tumor*.
- c. **Sebaceous horn:** Sometimes the contents of cyst are slowly discharged through the punctum and dry on skin surface to produce a sebaceous horn (Fig. 12.12).
- d. **Calcification:** It is a rare complication seen in long standing sebaceous cysts of scrotum.

- e. **Malignancy:** Very rarely, basal cell carcinoma may develop in a sebaceous cyst.

CYSTIC SWELLINGS FROM BLOOD VESSELS**Hemangioma**

It is a developmental malformation of blood vessels.

- It is an example of *hamartoma* (Fig. 12.13). The features of hamartoma are shown in Box 12.7.
- Hemangioma is commonly seen in skin and subcutaneous tissues of head and neck region but can occur in any part of the body.
- Types are *capillary*, *venous (cavernous)* and *arterial*. Their natural history is given in Box 12.8.



Fig. 12.13: Hamartoma forehead with vascular, lymphatic and fibrous elements

Capillary Hemangioma

It arises from capillary tissue. Its types are:

- Salmon patch:** It presents as a bluish patch on the forehead at birth. It disappears by the age of one year, hence treatment is reassurance only.
- Port-wine stain:** It is present since birth and usually does not show any change throughout life. It is commonly seen on face, neck and shoulders. It presents as a purple-red skin discoloration not raised above the surface. On pressure, the color blanches and reappears on release of pressure.

Indication for treatment is cosmetic disfigurement.

Various treatment modalities are:

- Cosmetics may be applied to mask the skin discoloration.
- Excision and skin grafting.
- Laser destruction of port-wine stain.

Sturge-Weber syndrome: When Port-wine stain of face is associated with hemangioma of ipsilateral cerebral hemisphere, epilepsy and glaucoma.

- Strawberry angioma:** It is commonly seen on face (Fig. 12.14). It involves skin, subcutaneous tissue,

Box 12.7: Features of hamartoma

- Developmental anomaly.
- Congenital in origin; mostly presents at birth.
- Normal tissue at normal place but in abnormal and excessive amount.
- Grows during childhood.
- Growth ceases with stoppage of body growth.
- May regress in size or even disappear spontaneously (e.g. strawberry hemangioma, Salmon patch).
- Essentially benign, does not invade or metastasize.
- Examples are:
Hemangioma (blood vessels)
Lymphangioma (Lymphatics)
Neurofibroma (Nerves)
Nevus (skin)



Fig. 12.14: Strawberry hemangioma forehead

and mucosa. The baby is normal at birth and a red mark is noticed at the age of one to three weeks. This rapidly increases in size up to three months of age and a strawberry like swelling is produced. It grows with the child up to age of one year. After that it starts fading gradually and usually disappears by the age of 7-8 years.

Box 12.8: Natural history of hemangiomas

Type of hemangioma	Presentation	Progress	Regression
Salmon patch	At birth	Static	Spontaneous at 1 year.
Portwine stain	At birth	Static	Does not regress spontaneously
Strawberry angioma	At 3 weeks	Increase up to 1 year	Spontaneous regression at 7-8 years
Cavernous angioma	At birth	Gradually increasing	Needs intervention (sclerotherapy, excision)
Arterial angioma	At birth	Gradually increasing	Needs intervention (embolization, excision)

On examination, it is a bright red or dark purple soft swelling raised from skin surface. The swelling is compressible and not pulsatile (cf. aneurysm).

Treatment:

- Watchful waiting till 7-8 years when natural involution occurs.
- Application of carbon dioxide snow.
- Injection of hypertonic saline, steroids or hot water.
- Excision with or without skin grafting.

Venous (Cavernous) Angioma

It consists of multiple dilated venous channels. It has no tendency to involute. It may rather become larger and troublesome with passage of time. Common sites are face, lips, ears and tongue. It presents as a bluish colored, soft swelling raised from the surface. The swelling is compressible but non-pulsatile.

The cavernous angioma is prone to ulceration and bleeding especially in oral cavity (Fig. 12.15).

Treatment

a. *Conservative treatment:*

- Intralesional injection of sclerosant (sodium tetradecyl sulphate, hypertonic saline).
- Application of Laser.
- If a feeding vessel is present, do therapeutic embolization. In this technique, a piece of gelfoam is injected into the feeding artery that causes ischemia and fibrosis of hemangioma.

b. *Surgical treatment:*

- Excision is better if swelling is small and localized.



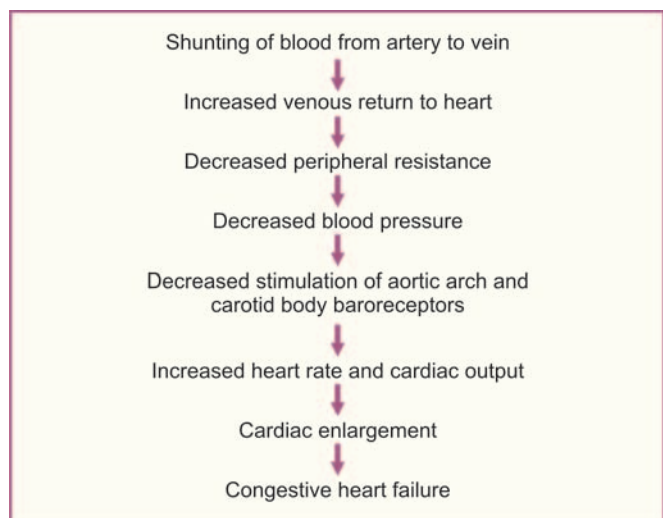
Fig. 12.15: Cavernous hemangioma tongue and lower lip

- If swelling is large and diffuse, it is better to shrink it in size by sclerotherapy before attempting excision.
- For excision of a big hemangioma in oral cavity, it is better to secure both external carotid arteries in neck beforehand so as to minimize bleeding.
- Diathermy is useful in controlling hemorrhage.

Arterial (Plexiform) Angioma

- It is congenital arteriovenous fistula.
- The AV fistula can also be acquired, e.g.
 - ☐ Following penetrating trauma.
 - ☐ Surgically created AV fistula in forearm for renal dialysis.
- The blood flows directly from artery to the vein and the vein becomes arterialized (dilated, tortuous and thick walled).
- The pathophysiological effects of AV fistula are shown in Box 12.9.
- *Clinical presentation* is as a:
 - ☐ Soft, pulsatile swelling.
 - ☐ Local temperature is slightly raised due to high vascularity.
 - ☐ Palpable thrill.
 - ☐ On auscultation, a continuous bruit (machinery murmur) is heard.
 - ☐ Overgrowth of affected limb may occur.
- *Nicolandi's or Branham's sign:* On compression of feeding artery swelling diminishes, the thrill and bruit disappear. The heart rate which is already increased due to AV fistula (See Box 12.9A) falls to near

Box 12.9A: Pathophysiology of AV fistula



Box 12.9B: Complications of AV fistula

- Ulceration
- Torrential hemorrhage (excessive, alarming bleed)
- Pressure on surrounding structures
- Congestive heart failure
- Hypertrophy of affected limb.

normal due to compression of feeding artery leading to decrease in venous return.

- *Complications* of AV fistula are shown in Box 12.9B.
- *Treatment:*
 - ☐ Angiography and selective embolization of feeding artery.
 - ☐ Surgical excision. Ligation of feeding vessels before surgical excision help in decreasing blood loss.

Cirroid Aneurysm

It is an AV fistula of the scalp vessels usually affecting temporal region in elderly people. The word 'aneurysm' is a misnomer. A soft, pulsatile, worm like swelling is felt in subcutaneous tissue in the temporal region. On auscultation, a bruit is heard.

Aneurysm

It is defined as localized dilatation of segment of an artery.

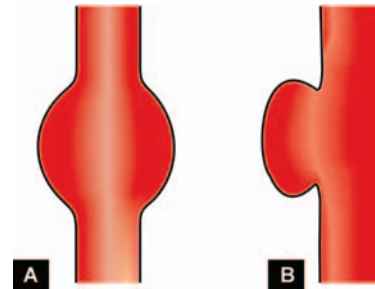
Aneurysm can involve large arteries like aorta, subclavian, carotid and femoral arteries or medium sized arteries like cerebral, renal and splenic arteries. Aneurysm of common carotid artery is atherosclerotic in origin and produces pulsatile neck swelling.

Etiology

- Atherosclerosis: Commonest cause.
- Traumatic.
- Congenital: Berry aneurysm in circle of Willis causing cerebral hemorrhage.
- Mycotic: Caused by bacterial infection and not the fungal infection. Hence, term 'mycotic' is a misnomer.
- Syphilitic.

Types

True aneurysm: Containing all three layers of arterial wall in aneurysmal sac.



Figs 12.16A and B: (A) Fusiform and (B) saccular aneurysm

False aneurysm: Containing fibrous tissue only in the wall of aneurysmal sac, e.g. following trauma.

Fusiform aneurysm: Segmental dilation of artery involving whole of its circumference (Fig. 12.16A).

Saccular aneurysm: Segmental dilation of artery involving part of its circumference (Fig. 12.16B).

Dissecting aneurysm: Due to defect in intima, blood tracks into the wall of the artery creating a false lumen. It usually involves aorta.

Clinical Features

- Smooth, cystic, pulsatile swelling in the course of artery. It shows 'expansile pulsations'.
- On proximal compression of artery, the swelling decreases in size and on releasing pressure it refills.
- A thrill is palpable over the swelling and on auscultation, a bruit is heard.
- The aneurysm may produce features due to pressure on adjoining structures, e.g. carotid artery aneurysm may press cervical sympathetic trunk producing Horner's syndrome (ptosis, myosis and enophthalmos).

Management

- Diagnosis is usually confirmed with CT angiography or MR angiography that shows extent of aneurysm.
- Small, asymptomatic aneurysms are treated conservatively.
- Large and symptomatic aneurysms are treated by resection of aneurysm with graft replacement (PTFE graft or Goretex graft).
- False aneurysm should always be treated surgically irrespective of its size.

CAROTID BODY TUMOR (CHEMODECTOMA) (SOLID SWELLING)

It is a rare tumor arising from chemoreceptor cells present on carotid bulb (at carotid bifurcation) (Box 12.10). The function of carotid body is regulation of pH. It is usually a benign tumor and rarely becomes malignant presenting with metastasis.

Higher incidence of carotid body tumor is seen in people living at high altitudes. It is possibly due to chronic hypoxia leading to carotid body hyperplasia.

Clinical Features

- It mostly presents in fifth decade.
- There is history of slow growing, painless lump in the neck for many years.
- The swelling is felt in anterior triangle of neck beneath anterior border of sternomastoid muscle at the level of 'Adam's apple' (Figs 12.17A and B).
- The swelling is firm, rubbery and compressible on firm pressure. It is shaped-like potato hence also called **potato tumor**.



Fig. 12.17A: Carotid body tumor right side

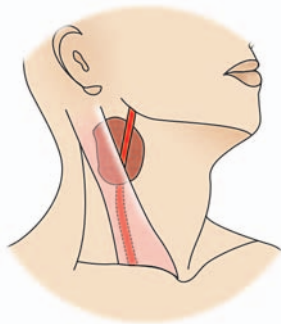


Fig. 12.17B: Diagrammatic representation of carotid body tumor

Box 12.10: Sites for chemoreceptor cells

- Carotid body receptors
- Aortic body receptors
- Myocardial receptors
- Pulmonary receptors
- Brainstem receptors

- The swelling is mobile side to side but not above downwards.
- The swelling is pulsatile. It gives transmitted pulsations because carotid artery is stretched over the swelling.
- On auscultation, a bruit can be heard over the swelling.
- Due to pressure effects of swelling the patient may present with Horner's syndrome (pressure on cervical sympathetic trunk) and hoarseness of voice (recurrent laryngeal nerve involvement).
- Some patients may present with a pharyngeal mass pushing the tonsil anteriorly and medially.

Investigations

- Duplex ultrasound demonstrates the location of tumor around carotid bifurcation.
- Carotid angiogram shows splaying of carotid bifurcation and blush of tumor vessels.
- FNAC/biopsy is contraindicated.

Treatment

- Elderly patients with asymptomatic tumor should not be operated since tumor is largely benign and complications of surgery are potentially serious.
- Large, symptomatic tumors are subjected to surgical excision.
- When tumor involves the carotid bifurcation; it requires resection of carotid artery with Dacron graft. In such cases, cerebral circulation has to be maintained with a bypass during the procedure to prevent cerebral ischemia.

STERNOMASTOID TUMOR (SOLID SWELLING)

It is due to trauma to the sternomastoid muscle during birth leading to a hematoma formation. The hematoma resolves with fibrosis leading to muscle shortening and formation of a swelling in the muscle. Hence, the term 'tumor' is a misnomer.



Fig. 12.18A: Sternomastoid tumor left side

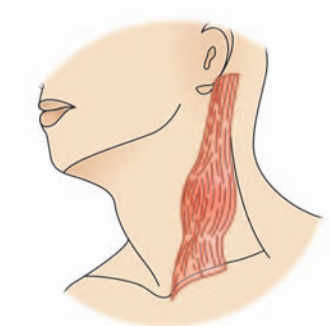


Fig. 12.18B: Diagrammatic representation of sternomastoid tumor

Box 12.11: Torticollis—causes

- Muscular: Contracture of sternomastoid
- Cervical: painful condition of cervical spine
- Pharyngeal: Infections, e.g. tonsillitis
- Ocular: Squint
- Intracranial: Posterior fossa tumor
- Postural

It usually presents in infants and young children. On examination, the affected sternomastoid muscle is stretched and chin is deviated to the opposite side (*Torticollis* or *Wry neck*) (Figs 12.18A and B). Various other causes of torticollis are given in Box 12.11.

A firm to hard swelling is felt in the affected muscle that is mobile side to side.

The lesion usually resolves without long-term effects. Physiotherapy to stretch the shortened sternomastoid muscle helps in most of the cases.

In severe cases, surgical release of the contracture is required.

CYSTIC SWELLING FROM LYMPHATICS

Lymphangioma

It is a developmental malformation (hamartoma) affecting lymphatics. Primitive lymph sacs develop during sixth week of intrauterine life. Failure of a part of lymph sac to join the main lymphatic system or sequestration of a portion of jugular sac results in a lymphangioma.

The common sites for lymphangioma are:

- Neck
- Axilla
- Groin
- Mediastinum
- Retroperitoneum

In the neck, it is called as *cystic hygroma* (Figs 12.19A and B). *Cystic hygroma* is a multilocular swelling consisting of multiple cysts filled with clear lymph and lined by a single layer of endothelium. These cysts are of variable size, intercommunicating with each other and may extend between muscle planes.



Fig. 12.19A: Cystic hygroma neck

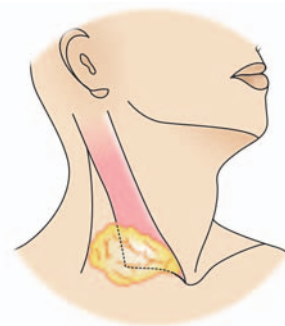


Fig. 12.19B: Diagrammatic representation of cystic hygroma neck

The cystic hygroma usually presents at time of birth or in early infancy. It may cause obstructed labor due to its large size. The location of swelling is in lower third of neck in the posterior triangle. The size of swelling may vary from small cystic mass to a huge lump occupying whole of the posterior triangle of neck extending up to cheek and ear.

On examination, the swelling is soft, cystic and partially compressible due to intercommunication of cystic spaces.

The swelling may increase in size on coughing or crying if there is intrathoracic extension. Most characteristic feature that distinguishes it from other similar swellings in the neck is that it is '*brilliantly transilluminant*'.

Complications

- Secondary infection* leading to painful swelling and fever. It may sometimes cause spontaneous regression of the lesion.
- Rapid enlargement* of cystic hygroma may cause respiratory obstruction in infants. It may require urgent aspiration of the cyst and even tracheostomy.

Treatment: Surgical excision of all the cysts and lymphatic tissues with preservation of normal neurovascular structures.

Injection sclerotherapy was earlier used for reducing the cyst size. However, it is not as effective since the lesion is multilocular. Moreover, it destroys the adjoining normal tissues and makes dissection more difficult.

Differential diagnosis: Box 12.12.

Box 12.12: Differential diagnosis of cystic swellings in posterior triangle of neck

Cystic hygroma	Lipoma	Hemangioma
Brilliantly transilluminant	Transillumination -ve	Transillumination -ve
Partly compressible	Non-compressible	Compressible
No skin discoloration	No skin discoloration	Skin discoloration +ve
Slip sign -ve	Slip sign +ve	Slip sign -ve
Non-pulsatile	Non-pulsatile	Can be pulsatile (AV fistula)

BRANCHIAL CYST

Embryology

During intrauterine life in the fifth week, four branchial arches are seen on the side of neck and grooves in

between are called as branchial clefts. The first cleft persists as external auditory canal. The second branchial arch overgrows and fuses with fourth arch thus obliterating the remaining three clefts. This potential space is known as "sinus of His" and persistence of this space results in development of branchial cyst.

Pathology

The cyst is lined by squamous epithelium. It contains thick turbid 'cheesy material' full of cholesterol crystals that is secreted by sebaceous glands in lining epithelium.

Clinical Features

- Although congenital, the cyst appears at 20-25 years of age because the fluid accumulates very slowly.
- There is painless swelling in anterior triangle of neck deep to sternomastoid muscle. The swelling is located at junction of upper and middle third of sternomastoid muscle bulging through its anterior border (Figs 12.20A and B).



Fig. 12.20A: Branchial cyst left side

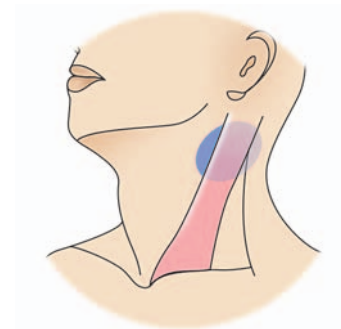


Fig.12.20B: Diagrammatic representation of branchial cyst

- The swelling is smooth surfaced, soft, cystic and fluctuant.
- On contraction of sternomastoid muscle, the swelling becomes less prominent.
- The swelling is non-transilluminant.

Diagnosis

- It is essentially clinical.
- Ultrasound shows a cystic mass.
- Needle aspiration shows turbid fluid rich in cholesterol crystals.

Differential Diagnosis

- *Cold abscess* in the neck—secondary to tuberculous lymphadenitis. It has ill-defined margins. Multiple enlarged matted lymph nodes are palpable in the neck. Constitutional symptoms of tuberculosis like loss of weight, anorexia and evening rise of temperature may be present.
- *Lymphangioma* in the neck is usually seen in infants in posterior triangle and produces brilliantly transilluminant swelling.
- *Carotid body tumor*: It is seen in elderly patients deep to sternomastoid muscle as a solid and pulsatile swelling.
- *Plunging ranula*: It produces a swelling in the submandibular region that is transilluminant and bimanually palpable through oral cavity.

Complications

- Due to presence of lymphatic tissue in the wall, there can be recurrent *infection* in the cyst. The cyst becomes painful and exhibits signs of inflammation.
- *Rupture* of the cyst or incomplete excision may lead to formation of branchial sinus.
- *Branchiogenic carcinoma*: Very rarely, primary squamous cell carcinoma in the branchial cyst is reported. However, before making a diagnosis of this rare condition, possibility of metastasis in cervical lymph nodes from occult primary in head and neck region must be ruled out.

Treatment

Excision of the cyst is carried out through transverse skin crease incision along the Langer's lines. The posterior wall of cyst should be carefully dissected since it may

extend up to pharyngeal wall. Its incomplete excision can lead to recurrence. One should avoid injury to spinal accessory nerve and hypoglossal nerve during dissection.

BRANCHIAL FISTULA

It is usually *congenital* and occurs due to failure of fusion of second branchial arch with the fourth arch. The external opening is situated in lower third of neck at anterior border of sternomastoid muscle (Fig. 12.21). It can be unilateral or bilateral. The track passes up between external and internal carotid arteries. The internal opening is situated in the pharynx at the level of posterior pillar of the tonsil (Fig. 12.22). However, deep part of the fistula tract is usually fibrosed and ends blindly near pharyngeal wall. Hence, it is a 'sinus' and not 'fistula' in most of the cases.



Fig. 12.21: External opening of branchial fistula

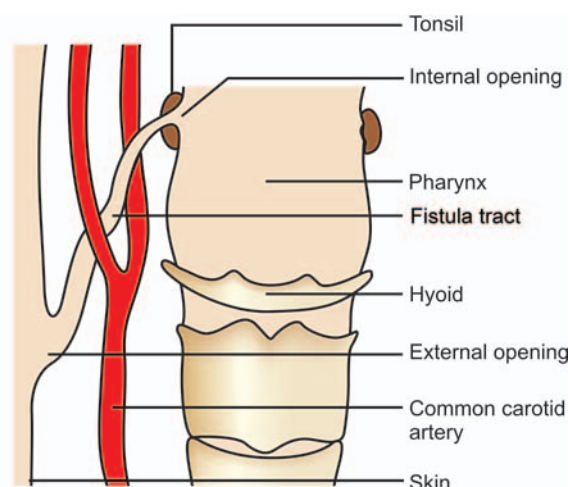


Fig. 12.22: Branchial fistula track

Sometimes, it can be *acquired* due to rupture of an infected branchial cyst. However, in such case, the skin opening is at higher level (junction of upper 1/3rd and middle 1/3rd of sternomastoid muscle).

The sinus track is lined by ciliated columnar epithelium and produces mucoid or mucopurulent discharge through external opening.

Differential Diagnosis

Thyroglossal fistula: It produces a fistulous opening in the midline of neck in relation to thyroid cartilage that moves with protrusion of tongue.

Treatment

Complete surgical excision of the tract. Following steps are followed during surgery:

- Transverse elliptical incision encircling external opening.
- The tract is dissected deep to platysma from below upwards as high as possible.



Fig. 12.23: Branchial fistula being explored through two parallel neck incisions

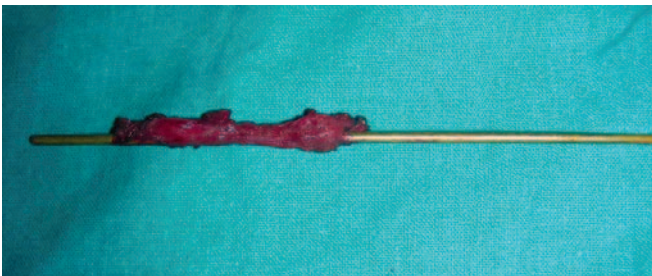


Fig. 12.24: Excised branchial fistula track mounted over a probe

- For further dissection, a second transverse skin incision is made at level of thyroid cartilage and dissected tract is taken out through second incision (Fig. 12.23).
- The fistula tract is dissected up to lateral pharyngeal wall where it is ligated and excised as a tubular track (Fig. 12.24).
- Both skin incisions are closed.

PHARYNGEAL POUCH

It is the protrusion of pharyngeal mucosa through a weak area in the posterior pharyngeal wall named as '*Killian's dehiscence*'.

Surgical Anatomy

At upper end of esophagus, a physiological sphincter is produced by inferior constrictor muscle. This muscle has two parts:

- Thyropharyngeus having oblique fibers.
- Cricopharyngeus having transverse fibers.

In between fibers of these two parts, there is a potential area of weakness called Killian's dehiscence.

If there is incomplete relaxation of inferior constrictor muscle during swallowing, it leads to rise in pharyngeal pressure and outpouching of mucosa through Killian's dehiscence (Fig. 12.25).

Clinical Features

- Mostly seen in elderly females.
- During early stage, there is difficulty in swallowing or foreign body sensation in the throat due to presence of small diverticulum.
- As diverticulum enlarged in size, patient complains of regurgitation of undigested food.
- There is recurrent chest infection due to aspiration from the pouch.
- In late cases, there is visible swelling in the neck behind sternomastoid muscle below the level of thyroid cartilage.
- On swallowing, the swelling increases in size and patient feels gurgling sound.
- On examination, swelling is smooth, soft with ill-defined margins. It gets reduced on pressure and is non-transilluminant.
- The patient has weight loss and cachexia due to starvation.

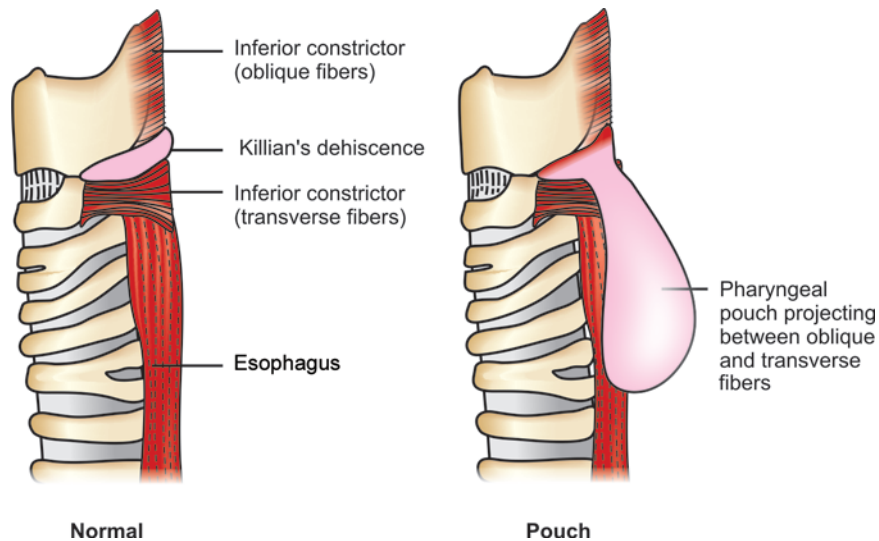


Fig. 12.25: Pharyngeal pouch—lateral view

Investigations

Barium swallow: Using thin barium in small amount to prevent aspiration pneumonia. It outlines the pouch.

Flexible esophagoscopy: Can show the opening of pouch. However, in unsuspected cases, there is risk of perforation of pouch during esophagoscopy.

Treatment

- In very old patients having early stage disease, treatment is conservative in form of chest physiotherapy, antibiotics for chest infection and nutritional support.
- In late cases, treatment is excision of pouch through a transverse skin incision. In all cases, cricopharyngeal myotomy is done to relax the sphincter.
- In recent years, endoscopic stapling technique is used and is found to be associated with high success rate and very low morbidity. In this technique, an endoscopic stapling gun is passed through oral cavity that safely divided the wall of pouch and adjoining cricopharyngeal muscle. It widens the neck of pouch and allows free drainage of pouch contents into esophagus.

LARYNGOCELE

- It is herniation of laryngeal mucosa through thyrohyoid membrane.
- It is seen in professional trumpet players, glass blowers and in patients with chronic cough.

- Patient may complain of hoarseness of voice due to displacement of vocal cords.
- It produces a narrow-necked swelling in the neck that contains air.
- The swelling is seen in the neck above thyroid cartilage. It becomes prominent when patient is asked to blow against closed mouth and nose (Valsalva maneuver).
- The swelling moves up on deglutition.
- On palpation, swelling is smooth, boggy and reducible. Cough impulse is present.
- The swelling is resonant on percussion.
- Secondary infection may occur leading to laryngo-pyocoele.
- Treatment is complete excision of sac with invagination of the stump.

SUBHYOID BURSAL CYST

- It is cystic swelling of the subhyoid bursa due to accumulation of inflammatory fluid.
- It is situated just below the hyoid bone over thyrohyoid membrane.
- The patient complains of painful swelling in the midline of neck.
- On examination there is a mildly tender, transversely elliptical (disc-shaped), cystic swelling below hyoid bone.
- It moves on deglutition due to attachment with hyoid bone.

- It does not move on protrusion of tongue (cf thyroglossal cyst).
- Treatment is surgical excision.

Parotid abscess: See Chapter 15—Diseases of Salivary Glands.

Retention cyst: See Chapter 15—Diseases of Salivary Glands.

Ranula: See Chapter 15—Diseases of Salivary Glands.

Cold abscess: See Chapter 4—Specific Infections.

CYSTICERCOSIS

- It is parasitic infestation by larval stage of tape worm (*Taenia solium*).
- Humans get infected by ingestion of cysticercus through undercooked pork or vegetables infected with larvae.
- Common sites of station are skeletal muscles and subcutaneous tissue.

Clinical Features

Involvement of CNS and eyes gives most serious manifestations in form of epilepsy and blindness respectively. In head and neck region, temporalis, masseter and sternomastoid muscles can be seat of involvement (Fig. 12.26). The cysts in muscle are usually asymptomatic and present with slow growing painless swelling.

On examination, a small, nontender, circumscribed and firm (tense cystic) swelling is felt in the affected muscle. On muscle contraction, the swelling becomes



Fig. 12.26: Tense cystic swelling right cheek becoming fixed on clenching teeth—cysticercosis masseter muscle

smaller in size and its mobility becomes restricted. The cyst may show signs of inflammation due to secondary bacterial infection.

Diagnosis

- *Serological tests:* ELISA and indirect hemagglutination tests.
- EITB (Enzyme-linked immunoelectrotransfer blot) assay is serological method of choice for diagnosis of neurocysticercosis.
- Ocular cysticercosis may be directly visualized on ophthalmoscopy.
- *Imaging:* X-ray may show spindle-shaped calcification in skeletal muscle.
- Ultrasound may show intramuscular cyst with nidus of parasite.
- CT and MRI are useful for diagnosis of neurocysticercosis.

Treatment

- Antiparasitic treatment with albendazole. Most neurocysticercosis are treated medically.
- Cysts in skeletal muscle and subcutaneous tissues can be excised.

MENINGOCELE

Due to failure of fusion of vertebral arch posteriorly, there is defect in vertebral column called as '*Spina bifida*'. The meninges protrude through this defect giving rise to a cystic swelling containing cerebrospinal fluid. It is entirely covered by healthy skin (Figs 12.27A and B).

Clinical Features

- Commonest site is lumbosacral region.
- It can also occur in occipital region and root of nose.
- It is present since birth.
- It is a hemispherical swelling.
- Sometimes swelling may be pedunculated or sessile.
- The swelling is tense, cystic, fluctuant and transilluminant.
- The swelling is compressible.
- An impulse is felt when the baby cries.
- A bony defect is felt at the margin of swelling.

Complications

- Ulceration



Fig. 12.27A: Cervical meningocele



Fig. 12.27B: Cervical meningocele (transilluminant swelling)

- Infection
- Rupture
- Hydrocephalus (Arnold-Chiari syndrome).

If the swelling contains spinal cord and nerve fibers along with CSF, then it is called *meningomyelocele*. In such cases, baby presents with neurological deficit that causes lower limb paralysis and bladder and bowel incontinence. On transillumination, the sac shows opaque bands due to presence of nerve fibers (Box 12.13).

Treatment

- The operation should be done within few hours after birth to minimize complications.

Box 12.13: Comparison between meningocele and meningomyelocele

	Meningocele	Meningomyelocele
Contents	CSF	CSF and nerve roots
Transillumination	Brilliant	Brilliant with nerve roots seen as opaque bands
Neurological deficit	Absent	Present
Prognosis after surgery	Good	Poor

- Excision of sac and closure of meninges is done.
- In meningomyelocele, nerve fibers should be carefully dissected and reposed back to minimize neurological deficit.
- In case of hydrocephalus, ventriculo-peritoneal shunting of CSF is done to decrease intracranial pressure.
- *Genetic counseling* of parents should be done for future pregnancy since there is 5% risk of having this condition in the offspring.

Myelocele

The spinal cord is exposed outside and its central canal opens to the exterior. The CSF dribbles constantly through the defect. The baby is usually stillborn and dies within a few days time.

ODONTOGENIC AND NON-ODONTOGENIC CYSTS

See Chapter 25—Swellings of the Jaws.

CLINICAL EXAMINATION AND DIFFERENTIAL DIAGNOSIS OF A NECK SWELLING

- See the location of swelling—anterior or posterior triangle.
- All midline swellings are included in anterior triangle swellings.
- In *anterior triangle swelling*, look for movement on swallowing. If it moves the possibilities are:
 - ☐ Thyroid swelling
 - ☐ Thyroglossal cyst
 - ☐ Subhyoid bursa
 - ☐ Laryngocele
 - ☐ Pretracheal lymph node

Now look for movement on protrusion of tongue. Only thyroglossal cyst moves on tongue protrusion.

- Then examine other characteristics of swelling as described in beginning of this chapter.
- The commonest swelling in the neck is lymph node swelling and enlarged lymph nodes are usually multiple.
- Whenever an enlarged lymph node is seen in the head and neck, always examine the drainage area to look for primary focus of infection/malignancy.
- Various causes of neck swellings in different parts of the neck are given in Boxes 12.14 to 12.18.

Box 12.14: Midline swellings neck

<i>Solid</i>	<i>Cystic</i>
Submental lymph node	Sublingual dermoid (non-transilluminant)
Pretracheal/ Prelaryngeal lymph node	Ranula (transilluminant)
Adenoma thyroid isthmus	Subhyoid bursitis (non-transilluminant)
Retrosternal goiter	Laryngocele (transilluminant)
Thymoma	Cold abscess in space of Burns (non-transilluminant)
Lipoma (in space of Burns)	
Chondroma (from manubrium sterni)	Innominate aneurysm (pulsatile)

Box 12.15: Swellings in anterior triangle neck

	<i>Solid</i>	<i>Cystic</i>
Submental triangle	Submental lymph node	Sublingual dermoid Ranula
Carotid triangle	Lymph node Carotid body tumor Sternomastoid tumor	Branchial cyst Cold abscess Carotid artery aneurysm
Submandibular triangle	Lymph node Submandibular salivary gland Tumor of mandible	Plunging ranula Lateral sublingual dermoid

Box 12.16: Swellings in posterior triangle of neck

<i>Solid</i>	<i>Cystic</i>
Lymph node	Cystic hygroma
Cervical rib	Cold abscess
Pancoast tumor*	Pharyngeal pouch Subclavian artery aneurysm Vertebral artery aneurysm
*Carcinoma in upper part of lung presenting as neck mass	

Box 12.17: Swellings in suprasternal space of Burns

- Lipoma (lobular swelling with slip sign)
- Dermoid (cystic swelling containing pultaceous material)
- Cold abscess (soft cystic swelling containing caseous matter with matted lymph nodes)
- Lymph node (firm, solid swelling, associated cervical lymph nodes enlarged)
- Innominate artery aneurysm (pulsatile)

Box 12.18: Swellings occurring anywhere in the neck

- Hemangioma
- Lipoma
- Neurofibroma
- Sebaceous cyst

13

Chapter

Diseases of Lymph Nodes and Lymphatics

Sanjay Marwah

CERVICAL LYMPH NODES

Surgical Anatomy

- There are about 800 lymph nodes in the body.
- Approximately 300 lymph nodes lie in the neck.
- The lymphatics of head and neck drain in cervical lymph nodes.
- Lymph nodes in neck are arranged in two groups:
 - i. *Superficial group*: These are present superficial to deep cervical fascia and are very few in number.
 - ii. *Deep group*: These are present deep-to-deep cervical fascia. They are further divided into two groups:
 - a. *Circular chain*: It consists of—
 - Submental
 - Submandibular
 - Preauricular
 - Postauricular
 - Occipital
 - b. *Vertical chain*: These glands lie in intimate relation to internal jugular vein and are deep to sternomastoid muscle. These are:

Jugulodigastric nodes: These lie below posterior belly of digastric muscle as it crosses internal jugular vein. These nodes drain nasopharynx, oropharynx, tonsils, posterior 1/3rd of tongue, upper larynx and pyriform fossae. These are the commonest site of involvement due to disease in these areas.

Jugulo-omohyoid nodes: These lie behind the mid part of internal jugular vein where it is crossed by anterior belly of omohyoid muscle. These nodes drain tongue, thyroid and mediastinal structures.

Supraclavicular nodes: These lie around inferior part of internal jugular vein and extend in the supraclavicular region. These nodes drain thyroid, esophagus, lungs and breast.

Virchow's lymph nodes: These are left supraclavicular group of lymph nodes lying between the two heads of sternomastoid muscle. These lymph nodes are enlarged due to metastasis from abdominal malignancies (stomach, colon, pancreas) and testicular tumors due to retrograde spread from thoracic duct (*Troisier's sign*).

Pretracheal and Paratracheal lymph nodes: Present around trachea and drain trachea and thyroid.

Adenoid tissue: This is lymphoid tissue present at the entrance of pharynx in a circular fashion and is known as *Waldeyer's lymphatic ring* (Fig. 13.1). It is formed by: Superiorly—adenoids in the roof of pharynx.

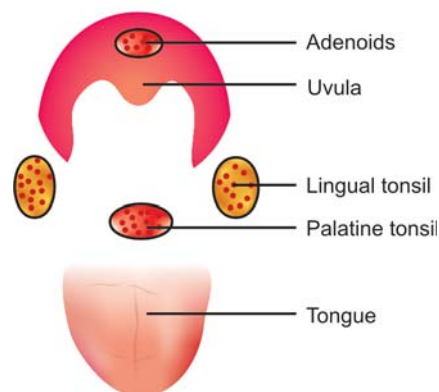


Fig. 13.1: Waldeyer's lymphatic ring

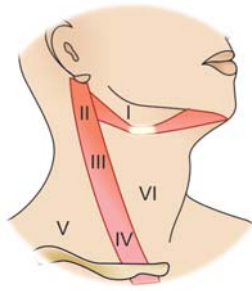


Fig. 13.2: Levels of lymph nodes in the neck

Inferiorly—lingual tonsils, i.e. lymphoid tissue at base of tongue.

Laterally—tonsils on side wall of pharynx.

- For neck dissection operations for lymph nodes, various levels of lymph node groups have been described for ease of identification of involved lymph nodes (Box 13.1A, Fig. 13.2).

Box 13.1A: Levels of lymph nodes in the neck

Level I	Submental and submandibular group.
Level II	Upper jugular group (Jugulodigastric)
Level III	Mid jugular group (Jugulo-omohyoid)
Level IV	Lower jugular group
Level V	Posterior triangle group
Level VI	Anterior compartment group (Prelaryngeal, Pretracheal, Paratracheal)

Clinical Examination of Lymph Nodes and Lymphatic System

History

- **History of swelling:** Ask following details
 - *Duration of swelling:* It is short in infective lymphadenitis (days) and long in metastatic lymph node deposits (few weeks or months) and tubercular lymphadenitis (months or years).
 - *Progress of swelling:* It is slow in tuberculosis and rapid in malignant deposits. Rapid increase in size in a day or two with pain and fever is suggestive of suppuration and abscess formation. There may be history of regression in size with antibiotic treatment in infective pathology while malignant deposits increase progressively.
 - *Pain in swelling:* Acute throbbing pain occurs in suppurative lymphadenitis. The lymph node enlargement in tuberculosis and malignancy is painless.

- *Other similar swellings:* These may appear at multiple sites (neck, axilla, groin) in generalized lymphadenopathy (lymphoma, tuberculosis).
- **History of fever:**
 - High grade fever of short duration occurs in acute infections.
 - Low grade fever with evening rise of temperature occurs in tuberculosis.
 - Remittent bouts of intermittent fever occur in lymphoma.
 - There is usually no fever in metastatic lymph nodes.
- **Weight loss:** If more than 10% of body weight is lost in six months time, it is considered as significant weight loss. It is seen in lymphoma, tuberculosis, malignancy.
- **Loss of appetite:** It is also seen in malignancy and tuberculosis.
- **History regarding site of primary pathology:**
 - Cervical lymph nodes appearing after dental sepsis are due to infective pathology.
 - Cervical lymph nodes appearing after non-healing ulcer in the tongue, hypersalivation, and disarticulation is suggestive of metastatic lymph nodes from carcinoma tongue.
- **Past history:** Ask about history of tuberculosis, exposure to sexually transmitted diseases (especially HIV) in the past.
- **Family history:** Ask about history of tuberculosis in family members.

General Physical Examination

- Anemia
- Jaundice
- Sternal tenderness (leukemia)
- Dilated veins in neck and chest (superior vena cava compression due to enlarged mediastinal nodes in lymphoma).
- Unilateral limb edema (arm edema in axillary nodes and pedal edema in inguinal nodes enlargement).

Local Examination

- In a patient presenting with cervical lymph node enlargement, remove clothing to expose neck, axillae and chest/breast.
- Inspection is done from the front to see the groups of enlarged lymph nodes. Look for associated lesion, e.g. tumors, sinuses, scars in head and neck region.



Fig. 13.3A: Method of palpating submandibular lymph nodes

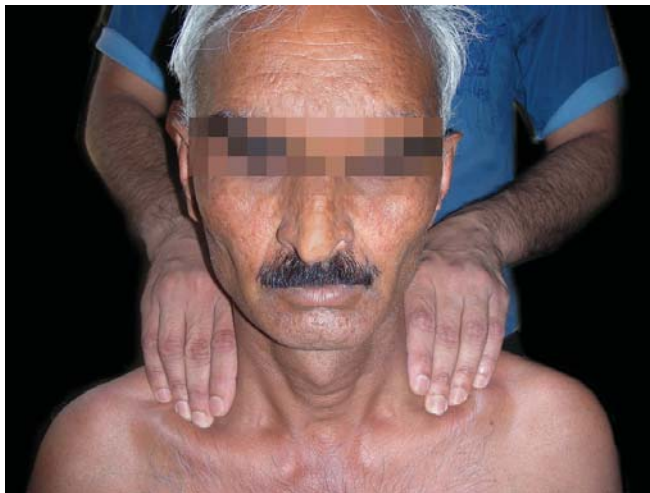


Fig. 13.3B: Method of palpating supraclavicular lymph nodes

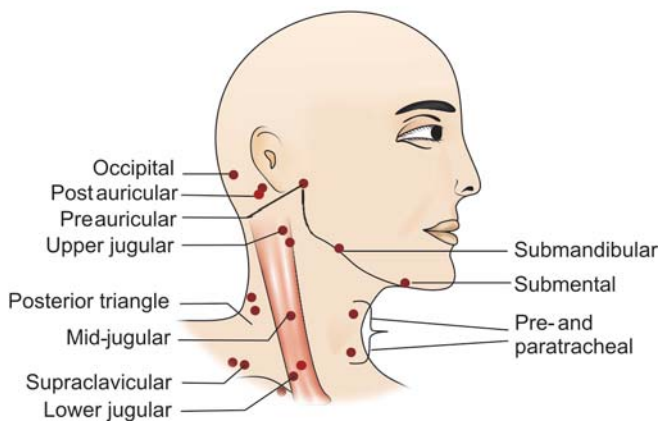


Fig. 13.4: Various groups of cervical lymph nodes

Box 13.1B: Findings on palpation of lymph nodes

- Site: More than two anatomical sites—generalized lymphadenopathy
- Number
- Size
- Surface
- Consistency:
 - Soft in acute infections
 - Firm in chronic infections (tuberculosis)
 - Hard in malignancy
 - Rubbery in lymphoma
- Fixity to skin: Fixed in malignancy, cold abscess
- Mobility on underlying structures:
 - Mobile in chronic infection
 - Fixed in infiltrating malignancy
- Matting: Tubercular lymphadenitis

- Palpation is best done by standing behind the patient (Figs 13.3A and B). Palpate all groups as depicted in Figure 13.4 and record findings (Box 13.1B).
- Flexion of neck helps in better palpation of submandibular nodes and vertical chain nodes.
- If any of the nodes are found enlarged, the corresponding drainage area is examined, i.e. scalp, ears, eyes, nose, oral cavity, face, neck, chest, etc. (Figs 13.5A to D).
- Oral cavity should be examined thoroughly using torch for illumination, tongue depressor for exposure and a gloved hand for intraoral palpation.
- Examine the other lymph node areas, e.g. axilla, groin, abdomen.
- Examine the abdomen for:
 - ☐ Hepatosplenomegaly (in lymphoma)
 - ☐ Any abdominal malignancy especially if left supraclavicular lymph nodes (Virchow's) are enlarged.
- Examine the testes for any tumor.
- Per-rectal and vaginal examination for any pelvic malignancy.

CASE SUMMARY

30 years old male presented with painful swelling in right submandibular region for the last 2 months. The diagnosis of cervical lymphadenitis was made and it responded to antibiotic treatment. However the swelling recurred after one month and developed an abscess as well (Fig. 13.5A). The case was referred for surgical opinion and oral cavity examination



Fig. 13.5A: Cervical lymphadenitis with overlying abscess



Fig. 13.5B: Oral cavity examination of the patient revealed dental sepsis as a cause of cervical lymphadenitis

revealed severe dental sepsis (Fig. 13.5B). Once dental sepsis was treated, the abscess as well as cervical lymphadenitis resolved completely.

Learning point—In patient presenting with cervical lymphadenitis, always examine the drainage area (head and neck) including oral cavity to look for the site of primary lesion.

Causes of Cervical Lymphadenopathy (Box 13.2)

Acute Suppurative Lymphadenitis

It is usually caused by bacterial infection. Common organisms are group A streptococci or staphylococci. Infection starts in throat and spreads to involve cervical lymph nodes.



Fig. 13.5C: Multiple hard lymph nodes in the neck of an old man



Fig. 13.5D: Examination of oral cavity of the old man revealed growth base of the tongue

Patient presents with fever, sore throat and enlarged tender lymph nodes.

Simple infection is treated with antibiotics (amoxycillin).

In case of abscess formation, it may require needle aspiration or incision and drainage.

Chronic Nonspecific Lymphadenitis

It is due to chronic infection in the drainage area, e.g. dental sepsis, recurrent tonsillitis, pediculosis capitis. It is also seen in cases of inadequate antibiotic treatment of infection in the drainage area. Upper deep cervical

Box 13.2: Causes of cervical lymphadenopathy**Infective**

- Acute suppurative lymphadenitis
- Chronic nonspecific lymphadenitis
- Tuberculous lymphadenitis
- Glandular fever
- Toxoplasmosis
- Cat scratch fever

Malignancy

- Secondary deposits
- Primary—Hodgkin's lymphoma
- Non-Hodgkin's lymphoma
- Burkitt's lymphoma
- Chronic lymphocytic leukemia

Autoimmune disease

- Systemic lupus erythematosus
- Juvenile rheumatoid arthritis.

lymph nodes are affected and involvement may be bilateral.

The lymph nodes are firm, mildly tender but not matted. FNAC of the cervical lymph node shows sinus histiocytosis or follicular hyperplasia. It helps in ruling out specific causes of lymphadenopathy.

Treatment is to treat the underlying cause and attend to the general health of the patient.

Tuberculous Lymphadenitis

Details given in Chapter 4: Specific Infections.

Glandular Fever (Infectious mononucleosis)

It is an acute viral infection caused by Epstein-Barr virus.

It usually affects teenagers.

Patient presents with fever, fatigue and sore throat. There is generalized lymphadenopathy, hepatosplenomegaly and skin rash.

The monospot test detects RBC agglutination by antibodies to EB virus.

Treatment is symptomatic.

Toxoplasmosis

It is caused by a protozoan, *Toxoplasma gondii*. It is transmitted by undercooked meat.

Patient presents with fever, myalgia and lymphadenopathy.

Cat Scratch Disease

There is history of contact with cats. Local inflammation occurs at site of injury. Two weeks later, regional lymph nodes become enlarged and acutely tender.

Lymph nodes often get suppurated containing sterile pus. The abscess subsides after drainage. It is a self-limiting disease. Antibiotics may be given in complex cases.

Secondary Deposits in Lymph Nodes

Any malignant tumor in head and neck region can metastasize to cervical lymph nodes. It is commonly seen in elderly individuals, usually males. However, metastasis from papillary carcinoma thyroid is seen in young adults.

The patient presents with painless enlargement of neck nodes.

There may be associated symptoms of primary lesion, e.g. sore throat, hoarseness, dysphagia, non-healing ulcer in oral cavity, cough, hemoptysis, etc.

The enlarged lymph nodes are stony hard, non-tender, mobile or fixed.

In elderly patients, greater cornu of hyoid bone is ossified and can be mistaken for a metastatic lymph node. However, on deglutition, the hyoid bone moves upwards.

Look for the evidence of primary growth in head and neck region.

If no primary growth is apparent, a specific search is made in oral cavity, nasopharynx, hypopharynx (nasopharyngoscopy), larynx (indirect laryngoscopy), external auditory canal, lung fields, breasts, chest wall and upper limbs. Various sites of *occult primary lesion* in metastatic lymph nodes are:

- Pyriform sinus
- Base of tongue
- Vallecula
- Nasopharynx
- Thyroid

In case of enlargement of Virchow's lymph node, look for abdominal malignancy (Troisier's sign) and testicular tumor.

When there is no evidence of primary lesion even after investigations, it is described as metastasis of unknown origin (MUO) (Fig. 13.6).

Staging of metastatic cervical lymph nodes: TNM classification of oral cancers (See Chapter 14: Diseases of Oral Cavity).



Fig. 13.6: Multiple hard lymph nodes in the neck with no evidence of primary tumor—MUO

Investigations

- Complete hemogram.
- *Triple endoscopy:* Direct laryngoscopy, bronchoscopy and esophagoscopy to look for any primary lesion.
- *Chest X-ray:* To look for primary or secondaries in the lungs, mediastinal lymph node enlargement.
- *X-ray paranasal sinuses:* For a tumor overlying the palate.
- *CT scan:* It is useful in detecting a small sized primary tumor, picks up small clinically impalpable lymph nodes and indicates extracapsular spread.
- *Biopsy of primary tumor.*
- If primary is occult, blind biopsies are taken from nasopharynx, pyriform sinus, base of tongue, tonsillar bed and esophagus.
- FNAC of enlarged cervical lymph node. Its accuracy is 98%. It can diagnose squamous cell carcinoma, adenocarcinoma and undifferentiated carcinoma.
- *Lymph node biopsy:* When aspiration cytology is inconclusive.

Operative Steps of Lymph Node Biopsy

The part is cleaned and draped. Local anesthesia is given by infiltrating 2% xylocaine. Skin incision is given directly over the lymph node along the skin crease. Skin, platysma and deep fascia are incised in line of incision. The lymph node is dissected out from surrounding tissues while holding it with Bebbcock forceps. In case,

there is a big matted lymph node mass adherent to vessels, a wedge biopsy of the mass is taken. Hemostasis is achieved. The fascia is closed with continuous chromic catgut sutures and the skin is closed with interrupted silk sutures or skin staples.

Treatment

- Surgical removal of primary lesion with en block dissection of lymph nodes.
- Radiotherapy to primary lesion as well as to lymph nodes.
- After radiotherapy, if primary tumor resolves and there are residual lymph nodes in neck, the nodes are removed by block dissection.

Metastatic Nodes—Secondary to Unknown Primary Tumor

The underlying pathology is diagnosed by FNAC or biopsy of involved lymph nodes.

- If histological diagnosis is squamous cell carcinoma and nodes are localized, consideration is given to block dissection of neck.
- If metastatic tumor is undifferentiated and nodes are large and multiple, primary site is presumed to be pharynx. Radiotherapy is given to pharynx along with nodes.
- Occasionally, it may be metastatic carcinoma from thyroid. It needs to be treated as thyroid carcinoma (Chapter 23: The Thyroid Gland).
- Sometimes it is metastatic adenocarcinoma suggestive of advanced abdominal malignancy.

Role of Chemotherapy

It is used in advanced head and neck cancers and aim is local control of disease.

Cisplatin and 5FU are the agents used.

Types of Neck Dissection

Radical Neck Dissection (Crile)

It involves resection of all lymph node groups from level I to level VI. The structures closely associated to lymph nodes are also removed. These are:

- Sternomastoid muscle
- Internal Jugular vein
- Accessory nerve

The main drawback of this surgery is cosmetic deformity and frozen shoulder due to paralysis of trapezius muscle (supplied by accessory nerve).

Modified Radical Neck Dissection

All cervical lymph nodes from level I to level VI are removed. However, one or more of the following three structures are preserved:

- Sternomastoid muscle
- Internal jugular vein
- Accessory nerve

Selective Neck Dissection

One or more of the major lymph node groups are preserved. Also sternomastoid muscle, internal jugular vein and accessory nerve are preserved, e.g. in *supraomohyoid neck dissection*, level I, II and III group of lymph nodes are removed. It is indicated in carcinoma of lower lip and floor of mouth. Its advantage is that both sides of neck can be operated at one operation.

Complications of neck dissection are given in Box 13.3.

Box 13.3: Complications of neck dissection

Immediate

- Hemorrhage
- Pneumothorax
- Raised intracranial pressure

Late

- Infection
- Chylous fistula
- Flap necrosis
- Carotid artery rupture
- Frozen shoulder

Hodgkin's Lymphoma

- It is a malignant tumor of lymphoreticular system arising mostly in lymph nodes and rarely in extra nodal sites (liver, spleen, etc.)
- It has bimodal age distribution (children and middle age people are mostly affected), more common in males (Box 13.4).
- It usually starts as painless enlargement of lymph nodes in left supraclavicular region (Fig. 13.7).



Fig.13.7: Large discrete rubbery lymph nodes in neck—Hodgkin's lymphoma

- Spread occurs to other lymph nodes in downstream lymphatic drainage in a systematic fashion.
- Grossly lymph nodes are pink-grey and cut surface is homogenous and smooth.
- Microscopically, characteristic **Reed-Sternberg cells** are seen. These are giant cells containing two large mirror image nuclei that may overlap (pennies on a plate appearance). In addition, histiocytes, plasma cells, eosinophils, lymphocytes, neutrophils may be seen. **“Cellular pleomorphism”** is a striking feature of Hodgkin's lymphoma.
- Depending upon type of cells, Hodgkin's lymphoma is divided into four types (*Rye classification*):
 1. *Lymphocytic predominant*: Plenty of mature lymphocytes and a few RS cells. It has excellent prognosis.
 2. *Nodular sclerosis*: Multiple thick bands of collagen tissue seen. It has good prognosis.
 3. *Mixed cellularity*: Mixed cell population seen. It has poor prognosis.
 4. *Lymphocytic depleted*: Very few lymphocytes and large number of RS cells seen. It is aggressive disease with poor prognosis.

Clinical Features

- Painless progressive enlargement of cervical lymph nodes.
- Following systemic symptoms may be present:
 - a. *Unexplained fever* with night sweats. Sometimes fever is seen in cyclical pattern (**Pel-Ebstein**

- fever**), i.e. fever for 5-7 days alternating with period of normal temperature of similar duration.
- b. *Unexplained weight loss*: 10% weight loss in six months duration is considered as significant.
 - c. *Pruritis*.
 - d. *Bone pains*: More after taking alcohol (seen in metastasis). Secondary deposits usually occur in lumbar vertebrae. These are osteosclerotic and pathological fracture rarely occurs.
- Sometimes patient presents with features of venous compression due to enlarged lymph nodes:
 - ☐ Dyspnea, hoarseness of voice, engorged neck veins due to superior vena cava compression.
 - ☐ Edema both legs due to inferior vena cava compression.
 - On local examination, cervical lymph nodes are nontender, smooth, rubbery and discrete (non-matted). Sometimes in late stages, matting of lymph nodes may be seen.
 - On general examination, there can be:
 - ☐ Anemia.
 - ☐ Jaundice.
 - ☐ Enlargement of other groups of lymph nodes.
 - ☐ Hepatosplenomegaly.
 - ☐ Pleural effusion.
 - ☐ Edema feet.
 - ☐ Red scaly patches of skin due to cutaneous involvement (**Mycosis Fungoides**).

Clinical Staging (Ann Arbor staging)

The aim of staging is:

- to determine extent of disease.
- to plan treatment.
- to assess the prognosis.

Stage I Confined to one lymph node site.

Stage II Involvement of more than one site, either all above or below the diaphragm.

Stage III Nodes involved above and below diaphragm.

Stage IV Involvement of extralymphatic sites, e.g. liver, bone, etc.

All stages are further subdivided into group A or B on the basis of absence (A) or presence (B) of the systemic symptoms such as weight loss, fever and night sweats.

Investigations

- Complete blood count with ESR to rule out leukemia.
- Renal function tests—blood urea, serum creatinine.
- Liver function tests.
- *Chest X-ray* to demonstrate enlarged mediastinal nodes and pleural effusion.
- Abdominal ultrasound to look for
 - ☐ Hepatosplenomegaly.
 - ☐ para-aortic lymph node enlargement.
- CT scan of the abdomen for better delineation of structures seen on ultrasound. Even small sized lymph nodes are picked up on CT scan.
- *Intravenous pyelography (IVP)*: To look for compression and back pressure effect (hydronephrosis) on kidneys due to enlarged para-aortic lymph nodes. However, if CECT abdomen is done, it clearly outlines kidneys and IVP is not required.
- *Bone scan*: If bony metastasis is suspected.
- *Bone marrow biopsy* may be required in case of hematological abnormality to look for bone marrow involvement.
- *FNAC*: It can give diagnosis of lymphoma. But histological pattern cannot be identified on FNAC.
- *Lymph node biopsy*: Excision biopsy is best for establishing the diagnosis and accurate histological grading.
- *Lower limb lymphangiography*: It can demonstrate pelvic and retroperitoneal nodes. A foamy or reticular appearance is characteristic of lymphoma. However, it is not done these days because of its invasive nature and availability of USG and CT scan.
- *Staging laparotomy*: It is also not done these days because of availability of CT scan and MRI that can detect early lesions. Earlier, it was done in clinical stage I, II and III of lymphoma. It helped in accurate staging of disease.

Steps

- ☐ Splenectomy (helps in downstaging the disease).
- ☐ Liver biopsy.
- ☐ Para-aortic lymph node biopsy.
- ☐ Mesenteric lymph node biopsy.
- ☐ Iliac crest marrow biopsy
- ☐ Oopharopaxy (in females).

Disadvantages

- ❑ Invasive procedure.
- ❑ Operative morbidity in form of pneumonitis, abdominal sepsis, wound infections, OPSI (overwhelming post-splenectomy infection).

Treatment

- *Radiotherapy (RT) and chemotherapy (CT)* are the two modes of treatment given according to stage of the disease.
- Both RT and CT are toxic and cause bone marrow depression. CT causes infertility in males.
- *Radiotherapy* is given in stage I, II and IIIa. A total dosage of 3500-4000 rads is given over a period of four weeks (five days a week). While giving radiotherapy, normal tissues are protected by lead shields. Various modes of delivery of RT are:

Involved field radiotherapy (IFRT): Only a specific group of involved lymph nodes is given RT.

Extended field radiotherapy (EFRT): It can be given as:

- a. *Mantle field RT:* It is given for supradiaphragmatic disease covering cervical, axillary and mediastinal nodes.
- b. *Inverted Y-field RT:* It is given for infradiaphragmatic disease covering para-aortic and iliac nodes.

Total axial nodal irradiation (TANI): It includes irradiation of both mantle and inverted Y-fields.

- *Chemotherapy:* It is given in stage IIIb and IV. Multiple cytotoxic drugs are given along with steroids to produce better remission. It is called as “combination chemotherapy”. Various regimens are:

MOPP: It includes

Mustine	6 mg/m ² I/V on Day 1 and Day 8.
hydrochloride	
Oncovin	1.4 mg/m ² I/V on Day 1 and Day 8.
Procarbazine	100 mg/m ² orally from Day 1 to Day 14.
Prednisolone	40 mg/m ² orally from Day 1 to Day 14.

- **ABVD:** It includes adriamycin, bleomycin, vinblastine and dacarbazine. It is less leukemogenic and causes less infertility.

Non-Hodgkin's Lymphoma

- It occurs in elderly age (60-80 years) (Box 13.4).
- Patient usually presents with generalized lymphadenopathy.
- Peripheral lymph nodes (centrifugal distribution) are more involved (e.g. epitrochlear lymph nodes).
- Waldeyer's ring is more commonly involved.
- Extranodal sites are more commonly involved, e.g. gut, bone marrow.
- Abdominal lymph nodes are more commonly involved and there is no definite pattern of lymph node involvement. So, there is no role of staging laparotomy in NHL.
- Systemic symptoms (B) are less common.
- The prognosis is poorer in comparison to Hodgkin's lymphoma.
- *Rappaport* has classified NHL into four morphological types each of which can be follicular or diffuse.
 1. Well-differentiated lymphocytic
 2. Poorly differentiated lymphocytic.
 3. Mixed lymphocytic and histiocytic.
 4. Histiocytic (Reticulum cell sarcoma).
- The prognosis is good in well-differentiated, small lymphocytic and follicular pattern (low grade tumors).
- The prognosis is poor in poorly differentiated, lymphoblastic and histiocytic pattern (high grade tumors).
- The prognosis is decided after lymph node biopsy showing histological pattern.

Treatment

Radiotherapy is given in stage I and II (low grade). Chemotherapy is given in stage I and II (high grade) and in stage III and IV.

Combination chemotherapy regimens are:

COP regimen:

Cyclophosphamide	600 mg /m ² I/V D ₁ and D ₈
Oncovin	1.4 mg /m ² I/V D ₁ and D ₈ .
Prednisolone	40 mg / m ² oral D ₁ to D ₁₄ .

CHOP regimen: It includes cyclophosphamide, hydroxy daunorubicin, oncovin and prednisolone. In case of failure of chemotherapy, whole body irradiation may be required.

Box 13.4: Hodgkin's vs non-Hodgkin's lymphoma

<i>Hodgkin's lymphoma</i>	<i>Non-Hodgkin's lymphoma</i>
i. Bimodal age.	i. Elderly age (60-80 years).
ii. Lymph nodes commonly involved—left supraclavicular.	ii. Abdominal lymph nodes.
iii. Waldeyer's ring—rarely involved.	iii. Commonly involved.
iv. Lymph node involvement—centripetal (mediastinal, para-aortic).	iv. Centrifugal (epitrochlear).
v. Extralymphatic sites—less involved.	v. More commonly involved.
vi. Pattern of spread in lymph nodes—Definite pattern, starting from cervical lymph nodes and then spreading downstream.	vi. No definite pattern of spread in lymph nodes.
vii. Staging laparotomy helpful.	vii. No role of staging laparotomy.
viii. Systemic symptoms (B)—more common.	viii. Less common.
ix. Microscopy—RS cell present.	ix. RS cells absent.
x. Prognosis—Good.	x. Poor.

Burkitt's Lymphoma

- Also known as malignant lymphoma of Africa. It is mostly seen in endemic areas of tropical Africa and New Guinea.
- EB virus that causes glandular fever is invariably found in Burkitt's lymphoma.
- High incidence in patients of AIDS.
- Young males between age of 3-12 years are more commonly affected.
- The child presents with soft, painless, rapidly growing jaw tumor or orbital tumor.
- The second commonest presentation is abdominal tumor (kidney, ovaries, GIT, retroperitoneal nodes involved).
- The patient may present with multiple, painless, raised reddish skin nodules.
- X-ray shows multiple small osteolytic lesions.
- Microscopic examination shows primitive lymphoid cells. **Starry night** is the characteristic appearance on low power microscopy.
- Treatment is radiotherapy and chemotherapy. Surgery is contraindicated because it is not curative and wounds fail to heal. Sometimes spontaneous remission may occur.
- Differential diagnosis is given in Box 13.5.

Leukemia

- Lymph node enlargement may be seen in most cases of lymphocytic leukemia (acute as well as chronic)

Box 13.5: Burkitt's lymphoma—differential diagnosis

- Soft tissue sarcoma
- Malignant melanoma
- T-cell lymphoma
- Metastatic skin deposits

and sometimes in chronic myeloid leukemia (during blast crisis).

- Chronic lymphocytic leukemia (CLL) is B-cell leukemia characterized by progressive accumulation of mature but functionally incompetent lymphocytes.
- Clinically, there is anemia, lymphadenopathy and hepatosplenomegaly.
- Investigations in a suspected case include PBF and bone marrow examination that show picture of leukemia. FNAC and lymph node biopsy show infiltration of lymph node by leukemic process.
- Chlorambucil is the mainstay of medical therapy in CLL.
- Bone marrow transplant is increasingly used in treatment.
- Splenectomy may help in cases of hypersplenism.

DISEASES OF LYMPHATIC SYSTEM**Anatomy of Lymphatic System**

- The lymphatic system develops from cystic spaces on either side of neck and groin. These large cystic spaces develop lymphatic vessels draining into them.

- Abdominal lymphatic channels drain into cisterna chyli present in the retroperitoneum. The thoracic duct originates from upper cisterna chyli just below the diaphragm, passes through posterior mediastinum and ends in left internal jugular vein in the neck.
- Lymphatics accompany veins everywhere except in cortical bones and central nervous system.

Physiology of Lymphatic System

- The main function of lymphatic system is to return lymph (protein rich fluid) from the interstitial space to back into circulation.
- About 3 liters of interstitial fluid is returned to circulation each day through lymphatics.
- The lymphatic system also allows lymphocytes to pass from lymph nodes to bloodstream.
- Lymphatic capillaries have large pores between endothelial cells that allow macromolecules to cross the wall.

Acute Lymphangitis

It is caused by *Streptococcus pyogenes* infection. It presents as reddish blue streaks in the skin involving area between the site of infection and draining lymph node group. Treatment is bed rest, limb elevation, antibiotics (cloxacillin).

Lymphedema

- It is excessive accumulation of tissue fluid in the extracellular space due to defective lymphatic drainage.
- Commonest site is lower limbs. Other sites are scrotum, penis and upper limbs.

Primary Lymphedema

The cause is unknown and considered to be congenital. Women are affected three times more than men. It is further subdivided into various types:

- On basis of age at presentation*
 - *Congenital*: It presents before 2 years of age. If it is familial, it is called as *Milroy's disease*.
 - *Praecox*: It presents at 2-35 years of age.
 - *Tarda*: It presents after 35 years of age.
- On basis of lymphangiographic findings*
 - *Aplasia*: There is complete absence of lymphatic trunks and the swelling is present from birth.

- *Hypoplasia*: The lymphatic trunks are fewer and smaller than usual. It is commonest variety. The swelling starts in early adult life after an attack of cellulitis.
- *Hyperplasia*: The lymphatics are enlarged, increased in number and tortuous (similar to varicose veins). The patient presents with discharging vesicles of milky fluid due to incompetent valves.

Secondary Lymphedema

It is much more common than primary form. There is destruction or obstruction of lymphatics due to some underlying cause.

- *Filariasis*: It is commonest cause of lymphedema worldwide. It is caused by *Wuchereria bancrofti* worm that enters the circulation by mosquito bite. It then enters the lymphatics and produces fibrotic inflammatory reaction in the lymph nodes. Initially, patient presents with high grade fever and chills, lymphangitis and epididymo-orchitis. Later, due to obstruction of lymphatic pathway, there is gross swelling of lower limb (*elephantiasis*) (Fig. 13.8). Hydrocele is a common manifestation.

The *diagnosis* is made by demonstration of microfilariae in peripheral blood film prepared during early morning.

Differential leukocyte count shows eosinophilia.

Complement fixation test may be positive.



Fig. 13.8: Elephantiasis left leg



Fig. 13.9: Lymphedema left arm and shoulder after mastectomy for carcinoma breast

Treatment is with diethyl carbamazine.

- **Malignant deposits:** It could be primary (lymphoma) or secondaries in the lymph nodes causing lymphatic obstruction.
- Following radiotherapy and surgical removal of regional lymph nodes for the treatment of cancer (most commonly of the breast) (Fig. 13.9).
- Trauma causing lymphatic disruption and venous thrombosis (e.g. degloving injuries).
- Chronic infections causing lymphangitis and lymphadenitis, e.g. tuberculosis.

Differential diagnosis of lymphedema: It is shown in Box 13.6.

Box: 13.6: Differential diagnosis of lymphedema

Cardiac failure	Bilateral
Renal failure	
Hepatic failure	
Hypoproteinemia	
Lipedema	
Myxoedema	Unilateral
Deep vein thrombosis	
Arteriovenous fistula	
Neurofibromatosis	
Diffuse lipomatosis	

Lipedema is bilateral symmetrical enlargement of legs due to deposition of abnormal fat. The feet are not involved. It almost exclusively affects women near puberty.

Clinical Features of Lymphedema

- Gradual swelling of one or both lower limbs.
- Limb size increases during the day and decreases at night but is never normal.
- Edema is pitting in early stage but becomes nonpitting in chronic stage due to subcutaneous tissue thickening.
- In long standing cases, skin becomes hyperkeratotic and fissured (pachydermatous appearance).
- Skin vesicles discharging milky fluid may be present.
- Skin infection in form of erythema and cellulitis may be present
- The patient should also be examined for:
 - ☐ Upper extremity lymphedema
 - ☐ Genital lymphedema
 - ☐ Hydrocele
 - ☐ Chylous ascites
 - ☐ Chylothorax.

Investigations

The diagnosis of lymphedema is essentially clinical. Investigations may be required to confirm the diagnosis in atypical and doubtful cases and to decide the type of surgical treatment.

Lymphangiography: Patent blue dye is injected in the web space to identify lymphatics. These lymphatics are cannulated and lipid soluble dye is injected into the lymphatics. The lymphatics are visualized as parallel tracks of uniform size that bifurcate as they proceed proximally. This test is 'gold standard' for showing structural abnormalities of larger lymphatics and lymph nodes. It is valuable if lymphatic bypass is considered. However, this test is technically difficult, may damage remaining lymphatics and requires general anesthesia. Hence, it has become obsolete as a routine method of investigation.

Isotope lymphoscintigraphy: It is most commonly used screening investigation and can be performed as out patient procedure. Radioactive technetium labeled colloid particles are injected subcutaneously in web space. These are taken up by lymphatics and pass

proximally to lymph nodes. Using gamma camera, radioactivity is measured at different time points. Proximal obstruction causes delay in progress of radioisotope.

CT scan and MRI imaging of the limb can help to differentiate lymphedema, venous edema and lipedema.

Pathological examination: FNAC or lymph node biopsy of enlarged lymph node can tell about underlying pathology (malignancy, tuberculosis, etc.).

Treatment

Conservative treatment:

- Limb elevation to reduce the edema.
- Graduated compression garments with maximum pressure at ankle and decreasing toward groin.
- Intermittent limb compression with pneumatic massaging device. It encourages interstitial fluid movement out of subcutaneous tissues.
- Weight reduction and exercise.
- Care of foot to prevent infections.
- Antibiotics for skin infections.
- *Benzpyrones* reduce edema by improving micro-circulation and exert anti-inflammatory effect.
- *Diuretics* have no role.

Surgical treatment: It is not indicated for cosmetic reasons. It is performed only in a few patients to improve functions.

- Bypass procedures:* These are performed in case of lymphatic obstruction seen on lymphangiography. The procedures can be:
 - Anastomosis between lymph node and vein.
 - Lymphovenous anastomosis.

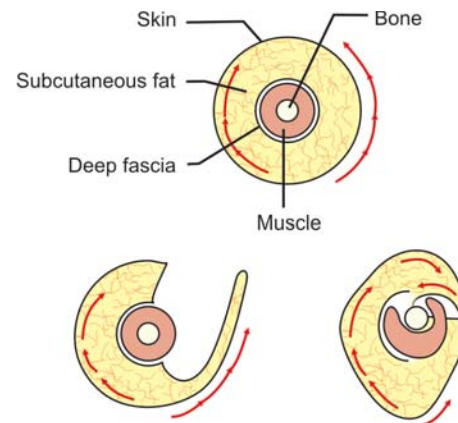


Fig. 13.10: Cut section image of Thompson procedure

b. Debulking procedures:

- *Sistrunk procedure:* A large wedge of skin and subcutaneous tissue is excised and the wound closed primarily. This procedure is no longer used.
- *Thompson procedure (Swiss roll operation):* Flaps of skin are de-epithelized and then buried in subfascial plane so that lymph will drain through skin lymphatics to deep fascial compartment. The procedure is largely abandoned due to poor results and complication of pilonidal sinus formation (Fig. 13.10).
- *Homans' procedure:* Skin flaps are raised, subcutaneous tissue is excised, flaps are trimmed and wound closed primarily. It can be performed only if skin is healthy.
- *Charles' procedure:* The skin and subcutaneous tissue are excised circumferentially down-to-deep fascia. Split skin grafts are then applied over the deep fascia. This procedure is useful in patients with unhealthy skin.

14

Chapter

Diseases of Oral Cavity

Sanjay Marwah

Following parts are included in the oral cavity:

- Mouth
- Tongue
- Lips
- Palate
- Tonsils

Oral cavity is limited anteriorly by lips, posteriorly by tonsils, laterally by cheeks, above by palate and below by floor of the mouth. Its lining epithelium is stratified squamous epithelium. Oral cavity suffers from various neoplastic and non-neoplastic lesions.

Various non-neoplastic lesions affecting different parts of the oral cavity are as follows:

DISEASES OF THE MOUTH

Stomatitis

It is an inflammatory condition affecting the mucous membrane of oral cavity leading to ulceration. Once ulcer forms in the oral cavity, it gets invaded by facultative organisms present in the oral cavity, viz. staphylococci, streptococci, *Borrelia vincentii*.

Predisposing factors for stomatitis are:

- Trauma due to sharp tooth, ill fitting denture or use of toothbrush with hard bristles.
- Protein energy malnutrition and deficiency of vitamins (vitamin B and vitamin C).
- Immunocompromised patients in conditions like:
 - ☐ Leukemia.
 - ☐ Aplastic anemia.
 - ☐ AIDS.
 - ☐ Patients receiving cytotoxic drugs, steroids for long duration.
 - ☐ Following radiotherapy for head and neck tumors.

- Autoimmune diseases like:

- ☐ Lichen planus.
- ☐ Behçet's disease.

- Chemicals like:

- ☐ Excessive ingestion of iodides.
- ☐ Lead, bismuth and mercury poisoning.

Various types of stomatitis are:

Aphthous Stomatitis

It is characterized by formation of a small, very painful ulcer in the oral cavity that is often associated with environmental or emotional stress. It commonly occurs on the lip, on the tip or sides of the tongue or mucosal lining of the cheek. It is small, round to oval in shape, with white floor and red erythematous margin (Fig. 14.1). The ulcer can be single or multiple. It usually starts in early adult life, mostly affecting females, tends to recur for some years and is rare after 50 years of age.

Treatment is chlorhexidine mouthwash and vitamins (vitamin C and B complex). The ulcer usually heals in 7-14 days time.



Fig. 14.1: Aphthous ulcer in mucosa of the lip; patient had carcinoma breast and was given chemotherapy

Monilial Stomatitis (Thrush)

It is a fungal infection of the oral cavity caused by *Candida albicans*. It is seen in following situations:

- In debilitated infants.
- In patients suffering from chronic debilitating ailments.
- In diabetic patients.
- In patients taking prolonged antibiotics or steroids.

Clinical features: The lesion starts as red spots on buccal mucosa that soon turns curdy white in appearance. The lesion is very painful and there is excessive salivation (Box 14.1). There can be painful swallowing due to involvement of pharynx.

Box 14.1: Ulcers in oral cavity

Painful	Painless
Aphthous ulcer	Malignant ulcer
Monilial ulcer	Syphilitic ulcer
Herpetic ulcer	SLE
Tubercular ulcer	Lichen planus
Dental ulcer	

Treatment:

- Chlorhexidine mouthwashes.
- Amphoterecin B lozenges.
- Nystatin cream locally.

Herpes Stomatitis

It is caused by herpes simplex viral infection. Most of the times this infection is subclinical and virus remains dormant. Reactivation of the virus occurs during febrile illness. The patient presents with fever and mucocutaneous lesions around the mouth involving lips, tongue, cheek and gums. Many small clear vesicles appear and soon breakdown to form yellow ulcers with bright red margins. The draining submandibular lymph nodes are enlarged.

Treatment includes plenty of fluids, soft diet, analgesics and antipyretics, topical application of acyclovir ointment.

Herpes zoster infection can sometimes occur as a result of reactivation of latent viral infection in immunocompromised patients. The patient presents with intraoral bullae and painful ulceration. Treatment is systemic antiviral therapy.

Angular Stomatitis (Angular cheilosis)

There is brownish superficial ulceration at the angle of mouth with scabbing that is often licked off by the patient

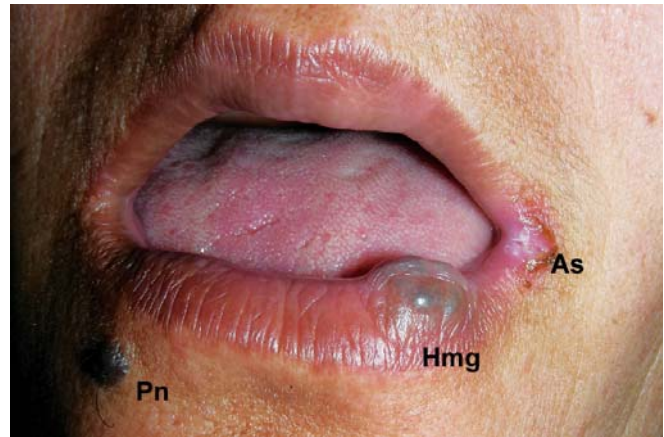


Fig. 14.2: Angular stomatitis (As); hemangioma (Hmg) and pigmented nevus (Pn) on lower lip are also seen

(Fig. 14.2). It is because of leak of saliva at the corner of mouth resulting in moist skin that gets ulcerated and infected by staphylococci or *Candida*. Its causes are:

- Children having habit of licking the corners of mouth (**perleche**).
- Elderly or edentulous patients with formation of skin creases at angle of mouth.
- Allergy to dentures or to lipstick.
- Vitamin deficiency (ariboflavinosis).

The most important *differential diagnosis* is syphilitic involvement of angle of mouth. In syphilis, fissuring is deeper, extends on to the mucous membrane and leaves permanent scars (Rhagades). However, angular stomatitis does not extend on to the mucous membrane and heals without scarring.

Treatment:

- Correction of denture.
- Improvement in general hygiene.
- Vitamin supplement.
- Miconazole cream for local infection.

Ulcerative Stomatitis (Vincent's angina)

- It is caused by *Borellia vincentii* (an anaerobic gram negative spirochete) and *B. fusiformis* (gram negative rod) present as normal commensals in oral cavity.
- It is precipitated by diabetes, stress, caries teeth and winter season.
- It is commonly seen in children and young adults.
- It does not affect the edentulous mouth.
- The patient presents with fever, malaise, painful gums, hypersalivation, foul smelling breath and painful swallowing.

- On examination, the gums are swollen, red, with or without ulcers covered with yellowish slough. The ulcers bleed readily and spontaneously.
- Once tonsillar infection occurs, it is called as Vincent's angina.

Treatment:

- Injection C penicillin 10 lac I/M 6 hourly for 7 days is the treatment of choice.
- Repeated mouthwashes with hydrogen peroxide.
- Improve nutrition.
- Dental treatment after acute attack subsides.

Gangrenous Stomatitis (cancrum oris)

See Chapter 3: Infections.

Syphilis

All three stages of syphilis can affect the mouth.

Primary syphilis: Chancre on lip or tongue.

Secondary syphilis: Snail track ulcers in mouth.

Tertiary syphilis: Gumma and chronic superficial glossitis. The latter is characterized by fissured tongue and loss of papillae. It is premalignant condition.

Congenital syphilis: Rhagades, Hutchinson's teeth, palatal perforation.

Details of syphilis are given in Chapter 4: Specific Infections.

Solitary Oral Ulcer

Its causes are:

- Traumatic—sharp tooth, denture, toothbrush injury.
- Malignant neoplasm.
- Tuberculosis.
- Syphilis.
- Fungal infection.
- Wegner's granulomatosis.
- Eosinophilic granuloma.
- Reticulum cell sarcoma.

If the cause is recurrent trauma, ulcer heals once source of irritation is removed. If ulcer still persists, it requires biopsy from the margin and treatment accordingly.

Cysts in the Mouth

- Mucus retention cyst:* See Chapter 15: Salivary Glands
- Sublingual dermoid cyst:* See Chapter 12: Cyst and Neck Swellings.

Submucous Fibrosis

- Due to deposition of collagen in submucous tissue, there is mottling and pallor of oral mucosa involving cheeks, tongue, palate and gums.
- There is restricted opening of mouth due to fibrosis leading to limited jaw movements.
- The lips and cheek become stiff.
- The condition is caused by chewing betel nut (Pan) and hypersensitivity to chilly.
- It is premalignant and may change to squamous cell carcinoma.

DISEASES OF THE TONGUE

Developmental Diseases

Congenital Fissuring of the Tongue

It presents at the age of 3-4 years and persists for life. The fissures of varying depth run transversely and the tongue surface is covered with normal papillae.

Differential diagnosis:

- Syphilitic tongue:* The fissures run in longitudinal direction and tongue is bald due to papillary atrophy.
- Fissuring of Ariboflavinosis:* The fissures run in longitudinal direction but are very deep and bottom of the fissures look 'beefy red'. There is associated angular stomatitis.

Tongue Tie

The lingual frenum is short and thick. It is revealed when the patient is asked to move the tongue upwards or outwards. It results in eversion of lateral margins and heaping up of middle portion of the tongue. It does not cause any disability. Treatment is division of the frenum with a scissor under local anesthesia. The resultant wound is closed in vertical direction. If done improperly, it can worsen the condition.

Lingual Thyroid

See Chapter 23: The Thyroid Gland.

Median Rhomboid Glossitis

There is formation of an ovoid or rhomboid mass in the midline posteriorly immediately in front of foramen cecum. It is slightly raised, devoid of papillae and appears distinct from the adjoining normal surface of the tongue. On palpation, the area is slightly indurated. Chronic infection by *Candida albicans* is not uncommon. It can be mistaken as a carcinoma.

Macroglossia

It means enlargement of tongue. It may not be obvious until patient protrudes out the tongue. Its causes are:

Developmental causes:

- Hamartoma
- Neurofibroma
- Lymphangioma

Acquired causes:

Cretinism: Prolonged hypothyroidism causes accumulation of mucoproteinaceous material.

Amyloidosis: There is deposition of amyloid in the tongue.

Acromegaly: Lips and nose are also enlarged along with tongue due to muscle hypertrophy.

Treatment: Treatment is of underlying cause in acquired cases. In developmental causes, the lesion needs to be excised. Elongation of tongue may be corrected by wedge excision of tongue. However, care should be taken not to injury nerve supply or lingual artery.

Geographic Tongue (Glossitis migrans)

Small bright red colored patches develop on the tongue due to denuded epithelium. These are surrounded by yellowish-white border. The epithelial regeneration and denudation occurs rapidly and the pattern of patches changes within one to two days. The condition is painless and its exact etiology is unknown. It is commonly seen in patients following abdominal operation for peritonitis and in patients with congenital heart defects. The condition persists as long as the patient is seriously ill and then subsides spontaneously.

Lichen Planus

It is seen as bluish-white patch on the tongue and offers problem in differential diagnosis. It is almost always accompanied with similar lesions on wrists and shins.

Black or Hairy Tongue

There is papillary hypertrophy in the posterior part on dorsum of the tongue. Tiny black particles of fungus stick to this patch and give it hairy appearance. This condition is seen in patients taking prolonged antibiotics leading to elimination of normal bacterial flora and superadded fungal infection (*Aspergillus niger*) of oral cavity.

Treatment is vitamin supplements, maintenance of oral hygiene and local miconazole cream.

Laceration of Tongue

Being very vascular, there is brisk hemorrhage following laceration of the tongue. Its causes are:

- Traumatic fractures of the jaws.
- Tongue bite during epilepsy.
- Injury by sharp objects, e.g. fishbone.

The brisk hemorrhage due to injury to lingual artery is controlled by hooking the tongue forward with a finger and compressing it against mandible by putting fingers in oral cavity and thumb in the submental region. The laceration is sutured taking deep bites.

In unconscious patient, brisk hemorrhage from the tongue laceration can choke the airway and may need emergency tracheostomy.

Inflammatory Lesions of Tongue

- i. *Pyogenic infections* of the tongue are very rare. It may occur as a part of Ludwig's angina leading to cellulitis of sublingual space and inflammatory edema of the tongue. Acute inflammatory swelling of the tongue may occur following wasp sting.
- ii. *Angioneurotic edema* may affect the tongue. It is sudden in onset, lasts for a short duration and may obstruct the airway. Its cause is not known. Treatment is immediate insertion of nasopharyngeal airway or tracheostomy.
- iii. *Chronic infections*
 - Tuberculosis
 - Syphilis

Ulcers of the Tongue

Various causes are:

Aphthous Ulcer

These are seen as small painful ulcers on the tip, sides and undersurface of the tongue in its anterior part. See 'Aphthous stomatitis'.

Dental Ulcer

It occurs due to mechanical irritation caused by a jagged tooth or denture. It is very painful and occurs on lateral margin. It is elongated in shape with slough at base and surrounding area of erythema. It heals when the cause is removed.

Postpertussis Ulcer

It is seen in whooping cough. The ulcer occurs on lingual frenum and undersurface of tip of the tongue because tongue protrudes over the lower incisors during bout of cough.

Tubercular Ulcer

It is rare these days. It complicates advanced, untreated pulmonary or laryngeal tuberculosis. The ulcers are often multiple and occur on the tip, dorsum or sides of anterior third of tongue. The patient complains of severe pain, difficulty in mastication and articulation. The ulcer is irregular in shape with undermined margins. The floor has pale granulation tissue and thin slough.

Syphilitic Ulcer

- a. *Primary chancre*: It may rarely occur on the tongue and presents as pustule near the tip. It bursts to form a painless ulcer that is surrounded by indurated tissue. The submental and submandibular lymph nodes get enlarged.
- b. *Gummatous ulcer*: Gumma occurs in midline in anterior 2/3rd of the tongue. Due to endarteritis, necrosis of the gumma occurs leading to ulcer formation. It is painless and has punched out edges with wash leather slough on floor.

Malignant Ulcer

It is usually seen in elderly patients (>50 years). Commonest site is lateral margin. It presents as non-healing ulcer with everted margins and indurated base. The draining lymph nodes are enlarged and hard.

Chronic Nonspecific Ulcer

It is usually present in anterior 2/3rd of the tongue. There is no definite etiological factor. There is no sharp tooth and there is no history of trauma. It is not very painful and only moderately indurated. Biopsy helps in ruling out specific lesions like tuberculosis and malignancy.

DISEASES OF THE LIPS

Pigmented Lips

Multiple, brown to black, pigmented spots are seen on the lips, inside of cheeks and palate in *Peutz-Jegher's*

syndrome. There is associated intestinal polyposis. Similar pigmented spots are likely to be present on the lips of the relatives since it is a familial condition. In *Addison's disease*, pigmentation is seen on lips but the pattern is more uniform.

Cracked Lips

It is seen in:

- a. *Angular stomatitis*: Cracks at angles of the mouth.
- b. *Exposure to cold weather*: It causes drying of the lips and crack is usually seen in midline of lower lip. Treatment is application of vaseline based ointment.

Macrocheilia

It means enlargement of the lips.

Its causes are:

- a. Lymphangioma
- b. Hemangioma
- c. Chronic inflammation
- d. Acromegaly

Chancre of the Lip

Syphilitic chancre may present as a painless ulcer of the lip. It has dull red floor and indurated base (button-like). The regional lymph nodes are enlarged.

DISEASES OF THE PALATE

Palatal Perforations

Hole in the palate leads to leakage of air into nasal cavity during phonation. It gives a peculiar nasal voice that is explosive in nature. Causes of palatal perforation are:

- Incomplete closure of hole during repair of cleft palate
- Following removal of malignant neoplasm of palate.
- Necrosis resulting from radiotherapy for a palatal malignancy
- Syphilitic gumma (rare cause).

Treatment: After taking care of the underlying cause, the palatal perforation can be covered by a denture or repaired by flaps based on one or both palatine arteries.

Palatal Swellings

It can occur in midline or laterally.

Causes of *midline swelling* are:

- Nasopalatine cyst*: It forms a bony swelling behind upper central incisors (See Chapter 25: Swellings of the Jaws).
- Median cyst*: Developmental cyst producing a bony hard swelling in the midline of palate posterior to incisive canal.
- Epstein pearls*: A group of small white cysts arranged in shape of a diamond at the junction of hard and soft palate in infants. These disappear spontaneously.

Causes of *lateral swelling* are:

- Dental cyst*: It arises in relation to normally erupted and chronically infected tooth.
- Glabulomaxillary cyst*: It is a developmental cyst presenting as a bulge between lateral incisors and canine tooth.
- Palatal cyst*: It arises from palatal mucous gland.
- Dental abscess*: It forms in relation to a carious tooth and has intense throbbing pain and trismus.
- Tumor of ectopic salivary gland*: Palate is the most frequent site although tumor may be found anywhere in the mouth or pharynx. It is a tumor of low grade malignancy and may invade base of skull and may metastasize to regional lymph nodes, viscera and skeleton.
- Neurofibroma of greater palatine nerve.
- Squamous cell carcinoma of palatal mucosa.
- Malignant neoplasm of maxillary sinus invading palate.

DISEASES OF THE TONSILS

Tonsil is an ovoid shaped, subepithelial aggregate of lymphoid tissue situated in lateral wall of oropharynx between anterior and posterior pillar. It forms a part of Waldeyer's ring that acts as a barrier to bacterial invasion. The tonsil contains deep tortuous crypts and has exceptionally good blood supply.

Acute Tonsillitis

It is acute inflammation of tonsil commonly seen in children and young adults.

Causes

Bacterial infection: Hemolytic streptococci, staphylococci, pneumococci.

Viral infection: *Infectious mononucleosis*

Clinical features: The patient presents with sore throat, malaise, fever, dysphagia and sometimes referred ear-ache. On examination, the tonsils are swollen and erythematous. Yellow or white pustules are seen covering the crypt, hence named *follicular tonsillitis*.

A throat swab should be taken at the time of examination and bacteriological examination helps to rule out diphtheria.

Treatment

- Warm saline gargles to wash away the purulent secretions.
- Analgesics and antipyretics to relieve pain and fever.
- Phenoxymethyl penicillin (penicillin V) is given initially and most patients respond well. In case of no response, antibiotics are changed according to swab culture and sensitivity report.
- Most of the cases resolve in 5-7 days.

Complications

See Box 14.2.

Box 14.2: Complications of acute tonsillitis

- Chronic tonsillitis
- Peritonsillar abscess
- Parapharyngeal abscess.
- Retropharyngeal abscess
- Ludwig's angina
- Rheumatic fever
- Subacute bacterial endocarditis
- Acute glomerulonephritis

Chronic Tonsillitis

It usually results from repeated attacks of acute tonsillitis. The tonsils become indurated and adherent due to fibrosis. It provides a reservoir for infective organisms leading to recurrent infection.

The patient presents with recurrent sore throat, fever and dysphagia. On examination, the tonsils are small

but contain pus and debris. The tonsillar lymph nodes are enlarged.

Treatment

Conservative in form of nutrition, maintaining oral hygiene and course of antibiotics and analgesics.

If condition recurs or persists, treatment is tonsillectomy.

Tonsillectomy

Indications

One of the important indications is enlarged tonsils causing chronic upper respiratory tract obstruction and sleep apnea. The diagnosis is made after hospitalization and performing sleep studies in the patient to establish the exact site and extent of the problem. Various indications of tonsillectomy are given in Box 14.3A.

Box 14.3A: Indications of tonsillectomy

Absolute

- Chronic upper respiratory obstruction causing sleep apnea.
- Suspected tonsillar malignancy.

Relative

- Chronic tonsillitis.
- Recurrent acute tonsillitis.
- Systemic disease due to recurrent tonsillitis, e.g. rheumatic fever, glomerulonephritis.
- Peritonsillar abscess.

Tonsillectomy should not be done when tonsils are acutely inflamed.

Steps

It is done under general anesthesia. Patient lies supine with head extended. The mucosa is incised over anterior faucial pillar and tonsil separated from its bed by blunt dissection till its pedicle is defined. Then pedicle is severed using a wire snare. A swab is placed in tonsillar bed to apply pressure for a few minutes so as to control bleeding. The swab is removed and bleeding points identified and controlled with suture ligation or bipolar cautery.

Complications

See Box 14.3B.

Box 14.3B: Complications of tonsillectomy

- Hemorrhage
- Pain (dysphagia, otalgia)
- Airway obstruction
- Infection
- Aspiration pneumonia

Peritonsillar Abscess (Quinsy)

There is formation of abscess in peritonsillar region between capsule of tonsil and superior constrictor muscle. It mostly occurs as a complication of acute tonsillitis and associated with streptococcal infection. It is commonly seen in adult males.

Clinical Features

There is severe pain in tonsillar region radiating to the ear and side of the neck. The patient has severe trismus and foul smelling breath. General symptoms include high grade fever with malaise. The patient presents to the clinician with his head held forward and upwards with a handkerchief. He talks as if he has 'hot potato' in his mouth. For examination, installation of local anesthetic may be required in posterior nasal cavity so that trismus is relieved and patient can open his mouth. On examination, there is diffuse swelling of the soft palate just above the involved tonsil and uvula is displaced to the opposite side. There may be pus pointing at the summit of the swelling.

Treatment

Patient is hospitalized and intravenous fluids are given. Parenteral analgesics and antibiotics may resolve the lesion during early phase. If condition persists, treatment is trans-oral incision and drainage of the pus. Interval tonsillectomy should be done after 4-6 weeks.

Parapharyngeal Abscess

- It is very similar to quinsy except that abscess occupies lateral pharyngeal space.
- There is maximum swelling behind the posterior faucial pillar.
- There is little or no edema of the palate.

- There can be diffuse swelling in the neck behind angle of the jaw and parotid region.
- Treatment is transoral incision and drainage of the pus using a blunt instrument.

Retropharyngeal Abscess

It can be *acute* or *chronic*.

Acute Retropharyngeal Abscess

There is formation of abscess in retropharyngeal space lying between pharynx in front and prevertebral fascia behind. This space is completely divided in the midline by a strong fascial septum into a right and left compartment. Hence, abscess always occurs on one side of the midline.

Etiology: Most commonly seen in children less than 1 year of age due to suppuration of retropharyngeal lymph nodes. The infection starts from tonsils, oropharynx or nasopharynx. In adults, it is rare and is caused due to injury of posterior pharyngeal wall by a foreign body, e.g. fish bone.

Clinical features: Generalized malaise, neck rigidity, dysphagia, dribbling saliva and marked dyspnea.

The apex of abscess is opposite the glottis and interferes with deglutition and breathing. Hence, child is seen characteristically holding his head in full extension with mouth open for maintaining adequate airway. On examination, inspection of posterior pharyngeal wall shows gross swelling with pointing abscess. On palpation, cushion like projection is felt on posterior pharyngeal wall.

Treatment:

- Hospitalization and intravenous fluids
- Antibiotics and analgesics
- Incision and drainage of the abscess. During drainage of abscess, airway should be protected by placing the child in head down position.

Chronic Retropharyngeal Abscess

- It is mostly tubercular in nature and rarely seen these days.
- It is due to anterior extension of tuberculosis of cervical spine. Since the abscess lies behind the prevertebral fascia, consequently it occupies the midline.

- Another cause is tuberculosis of retropharyngeal lymph nodes. In this situation, abscess is in front of prevertebral fascia in the retropharyngeal space and hence, gives swelling on one side of the midline (c/f acute retropharyngeal abscess).
- Unlike acute retropharyngeal abscess, this condition is solely seen in adults and there is no dysphagia or dyspnea.
- Apart from retropharyngeal swelling seen through oral cavity, the abscess extends in the neck and patient presents with fullness behind sternomastoid muscle on one side of the neck.
- The cervical spine is unstable and its manipulation may lead to neurological deficit.
- X-ray cervical spine shows evidence of bone destruction.

Treatment:

- Antitubercular drugs.
- If abscess persists, it is drained through cervical incision anterior to sternomastoid muscle.
- The abscess should not be drained through transoral route otherwise it may lead to secondary infection.
- Sometimes surgery is required for decompression of spinal cord to prevent progressive neurological deficit.

Infectious Mononucleosis (Glandular Fever)

It is a viral infection caused by Epstein-Barr virus. However, similar clinical features can be due to toxoplasmosis or cytomegalovirus.

Clinical Features

- Commonly seen in young adults.
- High grade fever with malaise.
- Throat pain and dysphagia.
- Hypersalivation.
- Difficulty in breathing.
- On examination, the tonsils are enlarged, edematous with a creamy-gray exudate.
- The tonsils are symmetrically enlarged and may appear to meet in the midline.
- There is generalized lymphadenopathy with hepatosplenomegaly.

Diagnosis: It is confirmed with serological test showing positive Paul-Bunnell test.

Treatment

- Hydration.
- Analgesics and antipyretic drugs.
- Patient may need hospitalization in case of respiratory obstruction.
- Steroids may help in relieving respiratory obstruction.
- If airway obstruction persists, elective tracheostomy should be done.
- Antibiotics are of no value since it is viral infection.
- Emergency tonsillectomy is contraindicated.

NEOPLASTIC LESIONS OF ORAL CAVITY

Benign Neoplasms

- Papilloma
- Hemangioma—mostly cavernous hemangioma.
- Lymphangioma
- Neurofibroma
- Minor salivary gland tumors
- Fibroma

Malignant Neoplasms

- Majority of the neoplasms arise from oral mucosa and are *squamous cell carcinoma* (85%).
- In oral cavity, carcinoma has predilection for the following sites:
 - ☐ Lateral margin and undersurface of the tongue
 - ☐ Floor of mouth
 - ☐ Retromolar trigone
 - ☐ Buccal mucosa
- Majority of the oral carcinomas develop without previous mucosal changes.
- Next common oral malignancy is malignant tumor arising from minor salivary glands.
- Rare malignant tumors of oral cavity are:
 - ☐ Malignant melanoma
 - ☐ Lymphoma
 - ☐ Sarcoma
 - ☐ Metastatic deposits

Incidence

In India, oral carcinoma is the most common malignancy. It accounts for about 40% of all malignancies. Its high incidence in India is because of betel chewing, tobacco, use of cigarette smoking (especially reverse

smoking) and alcohol. Oral carcinoma is mostly seen in elderly population (>60 years of age).

Although more common in males, the incidence is rising in females due to increasing use of tobacco and alcohol consumption.

Oral carcinoma is a preventable disease and its incidence can be drastically reduced by abstinence from pan, tobacco and alcohol.

Prognosis

Despite advancements in treatment modalities, the survival rates have not improved. Five-year survival has remained around 55% over past several decades. Possible reasons for this are:

- High incidence of multiple primary cancers in upper aerodigestive tract. Thus, following treatment of oral cancer at one site, second or third cancer may appear elsewhere in oral cavity even after several years.
- By the time oropharyngeal carcinoma is diagnosed, it is often a systemic disease. Development of distant metastatic disease leads to mortality.

Premalignant Lesions in Oral Cavity

i. Leukoplakia

It is white patch in the mouth that cannot be scraped. It cannot be characterized clinically or pathologically as any other disease.

Etiology: The causative factors are described classically as **6 'S'**:

Smoking, Spirit (alcohol), Spices (tobacco), Sepsis, Sharp tooth, Syphilis.

Another etiological factor is **oral candidiasis**.

Pathology: The microscopic features are:

- **Hyperkeratosis** Thickening of stratum corneum.
- **Acanthosis:** Proliferation of prickle cell layer that causes widening and elongation of rete pegs.
- **Parakeratosis:** Cells are still immature when they reach the surface epithelium and cells still retain their nuclei.
- As disorganization becomes greater, it is called as **dyskeratosis**.
- Epithelial pearls may be seen in the dermis and this is called **carcinoma in situ**.

Clinical features:

- Leukoplakia is mostly seen in middle aged or elderly people.
- It has 5% risk of malignant transformation. The risk of malignant change increases with the age.
- The only symptom is appearance of white patches in the oral cavity, usually tongue.
- On examination, leukoplakia may be seen in following stages:
 Stage-I: Appearance of thin milky film.
 Stage-II: Mucosa looks like smooth paint.
 Stage-III: Mucosa looks like wrinkled paint.
 Stage-IV: There is appearance of warty projections and ulceration.
- Mostly lesions are soft. Appearance of induration and ulceration is suggestive of malignant change.

Treatment:

- Suspicious areas (indurated, nodular or ulcerated areas) should be biopsied to rule out malignancy.
- If there is severe dysplasia or carcinoma *in situ*, it should be excised surgically or with CO₂ laser.
- If there is mild to moderate dysplasia, patient should be put on regular follow-up.
- If alcohol and tobacco consumption is stopped, leukoplakia may disappear spontaneously in many cases.

ii. Erythroplakia

- It is a bright red velvety plaque in the oral cavity that cannot be characterized clinically or pathologically as any other disease.
- The lesion is irregular in outline but clearly demarcated from adjacent normal epithelium.
- It may coexist with areas of leukoplakia.
- Erythroplakia has much higher incidence of malignant change. So it must be completely excised and subjected to pathological examination.

iii. Chronic Hyperplastic Candidiasis

- There is formation of dense chalky plaque that is thicker and more opaque than leukoplakia.
- It is commonly seen at oral commissures with extension on to adjoining skin of the face.
- The lesion has risk of malignant change.
- Treatment is long-term use of antifungal agents (Nystatin, Amphoterecin, Miconazole).

iv. Oral Submucous Fibrosis

- It is a progressive disease in which fibrous bands form beneath the oral mucosa.
- The condition is almost entirely confined to Asian countries and is associated with squamous cell carcinoma.
- It is thought to occur due to hypersensitivity to tobacco (Gutka), betel nut and chilli.
- The oral mucosa appears mottled or marbled due to collagen deposition in the submucosa.
- Mouth opening as well as tongue movements become limited due to loss of tissue elasticity.
- Treatment is surgical excision and grafting.
- Any known etiological factor should be removed.

v. Syphilitic Glossitis

- Syphilitic infection causes interstitial glossitis with endarteritis that leads to mucosal atrophy.
- The tongue becomes more prone to leukoplakia and other irritants causing oral cancer.
- However, cancer may develop in syphilitic glossitis without development of leukoplakia.
- Its incidence has decreased with the development of good antibiotics.

vi. Sideropenic Dysphagia (Plummer-Vinson syndrome or Paterson-Kelly syndrome)

- It is seen in iron deficiency anemia that causes epithelial atrophy of oral mucosa.
- The atrophic mucosa becomes vulnerable to carcinogenic irritants similar to that of syphilitic glossitis.

Certain **conditions** that may be **associated with oral cancers** are:

- Oral lichen planus*: Especially erosive lichen planus on lateral border of tongue carries an increased risk of malignant change.
- Discoid lupus erythematosus*: There are circumscribed and elevated white patches on the labial mucosa adjacent to vermillion border that may undergo malignant change.
- Dyskeratosis congenita*: It is a syndrome characterized by oral leukoplakia, nail dystrophy and reticular atrophy of skin with pigmentation.

CLINICAL FEATURES OF ORAL CANCERS (BOX 14.4)

Box 14.4: Clinical features of oral cancers

- Unexplained tooth mobility.
- Non-healing of sockets after tooth extraction.
- Non-fitting of dentures with inflamed gums.
- Hypersalivation, foul smelling breath.
- Slurring of speech.
- White or red patch in oral cavity.
- Non-healing, indurated ulcer with everted margins.
- Oral lesion fixed to underlying tissues (bone) or to overlying skin.
- Cervical lymph node enlargement.

Oral cancers give rise to early symptoms and can be easily examined and diagnosed. However, almost half the patients present with late lesions due to various reasons (Box 14.5).

Box 14.5 : Causes of late presentation of oral cancers

- Oral cancers are painless to begin with.
- Patients are elderly and frail.
- Many patients wear dentures and are used to discomfort and ulceration in mouth.
- Clinicians are often unsuspicious of malignant oral lesions and treat them conservatively.

Carcinoma Tongue

- It is the commonest site of oral cancer.
- Early cases are almost symptomless.
- Carcinoma of the tongue most commonly involves lateral borders (Fig. 14.3) (Box 14.6A).

Box 14.6A: Carcinoma tongue—site of involvement

Lateral margins	50%
Posterior 1/3rd	25%
Anterior 2/3rd	20%
(ventral surface, tip)	
Dorsum	5%

- Grossly, carcinoma tongue may appear as:
 - ☐ Non-healing ulcer.
 - ☐ An indurated and raised plaque.
 - ☐ A warty growth.
 - ☐ A deep and infected fissure.
- Features of carcinoma posterior 1/3rd of tongue are summarized in Box 14.6B.



Fig. 14.3: Carcinoma tip of the tongue presenting as non-healing ulcer

Box 14.6B: Carcinoma tongue (posterior 1/3rd)

- Late presentation.
- Presents with dysphagia, hoarseness of voice.
- Easily missed on examination.
- Palpation with gloved finger reveals induration.
- Bilateral neck nodes enlarged due to crossing of lymphatics.
- Important site for unknown primary.
- Poor prognosis.

- The advanced cases of carcinoma tongue present with:
 - ☐ Pain in the tongue with radiation to ear due to lingual nerve involvement.
 - ☐ Hypersalivation and difficulty in swallowing due to partially fixed tongue.
 - ☐ Foetor (foul smelling breath) due to necrosis and infection in the growth.
 - ☐ Ankyloglossia and dysarticulation.
 - ☐ Cervical lymph node enlargement due to metastatic deposits.
- The causes of mortality in advanced cases are:
 - ☐ Aspiration pneumonia.
 - ☐ Malignant cachexia.
 - ☐ Hemorrhage due to erosion of lingual artery or internal carotid artery.

Carcinoma Floor of Mouth

- It is the second commonest site for the oral cancer.
- Floor of mouth is U-shaped area between lower alveolus and ventral surface of the tongue.



Fig. 14.4: Carcinoma floor of mouth causing ankyloglossia

- Commonest site is anterior segment of floor of mouth to one side of the midline (Fig. 14.4).
- It is more commonly associated with pre-existing leukoplakia.
- The patient presents with indurated mass that soon ulcerates.
- The tumor rapidly involves adjoining structures, viz. tongue, gums and mandible.
- Involvement of tongue produces slurring of speech.
- Submandibular and jugulodigastric lymph nodes are enlarged bilaterally.

Carcinoma Buccal Mucosa (Cheek)

- The buccal mucosa extends above downwards from upper alveolar ridge to the lower alveolar ridge and anteroposteriorly from commissure to retromolar region.
- Carcinoma of the buccal mucosa is very common in India due to tobacco chewing.
- Commonest sites are retromolar area and commissure.
- Grossly, it may appear as:
 - Non-healing ulcer with sloughed floor and everted margins (Fig. 14.5).
 - Infiltrative growth with invasion of overlying skin (orocutaneous fistula), tongue, floor of mouth and mandible.
 - Exophytic growth called as verrucous carcinoma (See Box 14.7).
- The patient may present with trismus due to involvement of pterygoid muscles and masseter.
- Severe jaw pain due to periostitis and tumor infiltration of the mandible.



Fig. 14.5: Carcinoma buccal mucosa

Box 14.7: Verrucous carcinoma

- Exophytic growth.
 - No induration or deep invasion.
 - Very slow growing.
 - Soft, white, velvety mass.
 - Lymph node metastasis is late.
 - Low grade squamous cell carcinoma.
- Enlargement of submandibular and upper deep cervical lymph nodes.

Carcinoma Gingiva and Lower Alveolar Ridge

- Carcinoma of lower alveolar ridge mainly involves premolar and molar regions.
- The patient presents with proliferative or ulcerative lesion on gum margin (Fig. 14.6).



Fig. 14.6: Carcinoma lower alveolar ridge

- There is history of non-healing sockets following tooth extraction or history of sudden difficulty in wearing dentures.
- Diagnosis is often delayed because it is mostly considered as inflammatory lesion.
- Upper deep cervical lymph nodes are enlarged due to metastatic deposits.

Carcinoma Upper Alveolar Ridge, Floor of Maxillary Antrum and Hard Palate

Carcinoma arising from all these three anatomical sites has common presentation. (See Chapter 25: Swellings of the Jaw: Carcinoma Maxillary Antrum).

Carcinoma Lip

- It is more common in western countries in elderly people.
- It is most common on vermillion border of lower lip in farmers due to exposure to sun light (Countryman's lip).
- The patient presents with nonhealing ulcer having everted margins, indurated base and bleeds on touching.
- The tumor initially spreads laterally and later it spreads deeply invading the mandible.
- Lymph node metastasis in the neck occurs late.
- Differential diagnosis is given in Box 14.8.

Box 14.8: Carcinoma lip—differential diagnosis

- Leukoplakia
- Dental ulcer
- Minor salivary gland tumor
- Keratoacanthoma
- Pyogenic granuloma

Carcinoma Tonsil

- It constitutes 85% malignant tumors of tonsil while lymphoma is the second commonest tumor of the tonsil.
- The patient is usually elderly male presenting with pain and lump in the throat. The pain is severe and radiates to the ear.
- There is foul smelling breath and thick speech.
- On examination, there is unilateral enlargement of tonsil with ulceration of overlying mucosa. The growth may extend to involve adjoining palate.

- Cervical lymph nodes are enlarged.
- Untreated cases develop dysphagia, dyspnea and bleeding leading to death.

INVESTIGATIONS FOR ORAL CANCERS

Early diagnosis of oral cancers depends on:

- Awareness of the patient.
- High index of suspicion by the doctor.
- Careful clinical examination.
- Early biopsy of the suspicious lesion.

The relevant investigations in a suspected case of oral cancer are:

- Biopsy*: Surgical biopsy from suspicious area in the oral cavity can be done under local anesthesia. The biopsy should be taken from the margin of the lesion taking some of the adjoining normal tissue as well and avoiding areas of necrosis.
- Fine needle aspiration cytology*: FNAC of the enlarged cervical lymph nodes helps in detecting metastatic deposits.
- Plain radiography*: Orthopantomography of the jaws is helpful in assessing the involvement of bones. However, it has limited value because at least 50% of bone must be destroyed before radiological changes appear. X-ray chest may help in detecting pulmonary metastasis.
- CT scan*: CT scan is better than plain radiography for evaluation of antral tumors because it is highly sensitive in detecting cortical bone destruction. It is useful in detecting metastatic deposits in cervical lymph nodes, lungs, liver and brain. It has limited value in evaluation of intraoral tumors.
- MRI*: It is the investigation of choice for oral cancers. It is better than CT scan in defining the extent of soft tissue tumor (tongue). Moreover, unlike CT scan, MRI images are not degraded by presence of artifacts (dental amalgams in the oral cavity). It is as good as CT scan in diagnosis of cervical lymph node metastasis.
- Ultrasound*: It is useful in detecting liver metastasis. It is also helpful in guiding FNAC of cervical lymph node metastasis.
- Lab investigations*: These are done to evaluate patient's fitness for surgery and to exclude medical illnesses.

Box 14.9A: TNM classification for oral cancers

Primary tumor			
	T ₁	< 2 cm	
	T ₂	> 2-4 cm	
	T ₃	> 4 cm	
	T ₄	Adjacent structures invaded	
Neck nodes			
	N ₁	Ipsilateral single node < 3 cm.	
	N _{2a}	Ipsilateral single node > 3-6 cm.	
	N _{2b}	Ipsilateral multiple nodes < 6 cm	
	N _{2c}	Bilateral or contralateral nodes < 6 cm	
	N ₃	Any node (s) > 6 cm	
Distant metastasis			
	M ₀	No detectable distant metastasis	
	M ₁	Distant metastasis present	
Stage grouping			
Stage I	T ₁	N ₀	M ₀
Stage II	T ₂	N ₀	M ₀
Stage III	T ₃	N ₀	M ₀
	T ₁	N ₁	M ₀
	T ₂	N ₁	M ₀
	T ₃	N ₁	M ₀
Stage IV a	T ₄	N ₀	M ₀
	T ₄	N ₁	M ₀
	Any T	N ₂	M ₀
Stage IV b	Any T	N ₃	M ₀
Stage IV c	Any T	Any N	M ₁

- Surgery or radiotherapy is used alone or in combination with or without chemotherapy.
- Factors deciding treatment options in a patient are:
 - a. *Site of lesion*: For tumors involving alveolar process and mandible, surgery is the preferred treatment. With radiotherapy, there is risk of osteoradionecrosis.
 - b. *Stage of disease*: Early stage disease (stage I/II) can be cured with surgery or radiotherapy alone. Advanced stage disease (stage III/IV) requires combined modality treatment of radical surgery and reconstruction followed by postoperative radiotherapy. Inoperable disease is managed by palliative radiotherapy and/or palliative chemotherapy.
 - c. *Associated oral lesions*: If there are associated premalignant oral lesions (e.g. submucous fibrosis), surgery is preferable to radiotherapy.
 - d. *Tumor histology*: Squamous cell carcinoma can be treated by surgery as well as radiotherapy. Melanoma and adenocarcinoma are radioresistant and are treated by surgery.
 - e. Patient's physical fitness for surgery.
 - f. Physician's experience and skill.
 - g. Availability of treatment modalities.
- Treatment of oral cancers can be divided into two groups:
 - a. Treatment of primary tumor.
 - b. Treatment of neck nodes.

STAGING OF ORAL CANCERS

- TNM staging of oral cancers is done after clinical examination and investigations (Box 14.9A).
- It helps in treatment planning and in assessing prognosis.
- However, it has following drawbacks:
 - ☐ It ignores depth of tumor invasion, level of lymph nodes and lymph node fixity. These are important prognostic factors.
 - ☐ Clinical examination of the neck may miss the nodal metastatic disease in many cases.
 - ☐ TNM staging does not help in deciding operability in a given patient.

TREATMENT OF ORAL CANCERS

The treatment modalities for oral cancers are:

Surgery
Radiotherapy
Chemotherapy

A. TREATMENT OF PRIMARY TUMOR**(i) SURGERY**

Various advantages and disadvantages of surgery are given in Box 14.9B.

Box 14.9B: Surgical resection of oral cancers**Advantages**

- Expeditious
- Cost effective
- Less long-term sequelae
- Repeated intervention is possible in multiple primary lesions.

Disadvantages

- Cosmetic deformity
- Functional disability
- Risk of anesthesia



Fig. 14.7: Wide excision for small tongue ulcer; wound left open to granulate

Carcinoma Tongue

- Surgery is the treatment of choice for early lesions.
- Small lesions on the lateral border of tongue are treated with **wedge excision** taking 2 cm tumor free margin. If less than 1/3rd of tongue is removed, then formal reconstruction is unnecessary. The defect is cauterized and allowed to granulate and epithelialize spontaneously (Fig. 14.7).
- If CO₂ laser is used for excision, it has advantage of minimal edema and pain in the postoperative period and healing occurs with minimal scarring.
- Lesion more than 2 cm requires a **hemiglossectomy** in which half of the tongue is removed on one side of the midline. The resulting defect is repaired with split thickness skin graft.
- Larger tumors may require **total glossectomy** that carries high morbidity and mortality.
- If carcinoma tongue is involving floor of the mouth and mandible, it requires **Commando's operation**. The tumor is accessed via lip split and mandibulotomy. A paramedian mandibulotomy is preferred over a midline mandibulotomy as it does not disturb the hyomandibular complex and preserves the ability to swallow. The procedure involves hemiglossectomy, hemimandibulectomy, removal of floor of mouth and radical neck dissection.
- Whenever possible, one of the hypoglossal nerve should be preserved so that patient can relearn to speak and swallow.
- **Mandibular resection:** Need to resect any part of the mandible depends upon the involvement of mandible and its proximity to the tumor. If the tumor is in close proximity to the lower gingiva or extends

on to the mandible without its clinical or radiological involvement, then **marginal mandibulectomy** should be done. The procedure involves discontinuity excision of the tumor with a margin of mandible and overlying gingiva. It has good cosmetic and functional result since mandibular continuity is maintained.

If the tumor is directly invading the bone, then **segmental** or **hemimandibulectomy** should be done. Resection of the posterior part of body or ramus of mandible leaves very little cosmetic deformity and some functional deformity. But resection of the anterior arch results in significant functional and cosmetic deformity (**Andy Gump deformity**). So, immediate reconstruction should be done.

- **Reconstruction:** Extensive defects following radical resection require reconstruction with distant flaps. The **radial forearm free flap** allows one stage reconstruction and is now considered as work horse of oral reconstruction.

The **pectoralis major myocutaneous flap** is based on acromiothoracic artery and vein. The flap consists of pectoralis major muscle and an island of overlying skin. The pedicle is brought up by tunneling under the skin of chest wall and neck and it easily reaches the oral cavity. Neck dissection including removal of sternocleidomastoid muscle is usually combined to create space for the flap (See Chapter 27: Burns and Skin Grafting).

Carcinoma Floor of Mouth

- It spreads early to involve tongue as well as lower alveolus.
- Small tumors are treated by simple excision with 1 cm tumor free margin and the defect can be left to granulate.
- Large tumors invading tongue and mandible require Commando's operation. Immediate primary reconstruction should be done to avoid cosmetic and functional deformity.

Carcinoma Buccal Mucosa

- Small lesions localized to buccal mucosa are treated by wide excision followed by primary closure or split skin grafting.
- For big lesions extending to adjoining structures (maxillary tuberosity, mandible, tonsillar fossa), wide

excision followed by flap reconstruction is done. The **flaps** used are:

- Buccal fat pad** as a local flap to cover small intra oral defect (3×5 cm).
- Free radial forearm flap** for larger defects.
- Forehead flap** based on superficial temporal artery has been used extensively in the past to cover intraoral defects. However, it is rarely used now due to following reasons:
 - ☐ Cosmetic defect at donor site (forehead).
 - ☐ Two stage procedure requiring division of the pedicle at three weeks.
 (See Chapter 27: Burns and Skin Grafting).

Carcinoma Lower Alveolus

- Carcinoma of the lower gingiva can be treated with **marginal mandibulectomy**.
- If there is extensive bone involvement, it requires **segmental resection** or **hemimandibulectomy**. The primary reconstruction of the defect is always done in following ways:
 - ☐ **Free radial forearm flap** with a section of radius bone to fill the bony defect.
 - ☐ **Compound groin flap** based on deep circumflex iliac vessels.
 - ☐ **Free fibular flap**.
 - ☐ **Titanium mesh trays** packed with cancellous bone from ilium can be used for mandibular reconstruction.

Carcinoma Hard Palate, Upper Alveolus and Floor of Maxillary Antrum

See treatment of malignant tumors of maxilla in Chapter 25: Swellings of the Jaw.

Carcinoma Lip

- Up to $1/3$ rd of the lower lip can be removed with **V-excision** followed by primary closure in three layers (mucosa, muscle and skin).
- If more than $1/3$ rd of the lip is removed, primary closure results in microstomia. Hence, flap reconstruction is needed as follows:
 - Abbe flap:** A pedicled flap based on upper labial artery is rotated down to fill the defect in lower lip (Fig. 14.8).
 - Estlander's flap:** A wedge-shaped flap taken from the upper lip based on upper labial artery is rotated down to fill the defect in lower lip (Fig. 14.9).
 - Johansen stepladder procedure:** It raises symmetrical bilateral flaps from the lower third

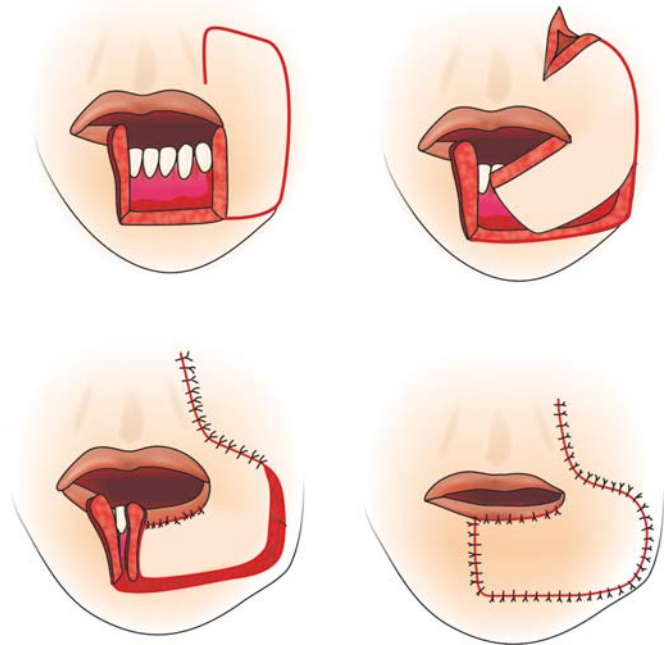


Fig. 14.8: Abbe flap

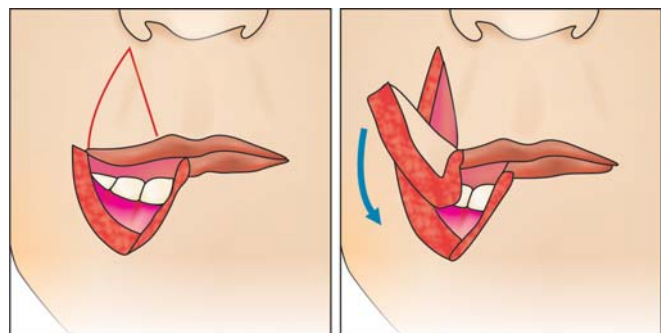


Fig. 14.9: Estlander's flap

of face to cover large central defect in the lower lip. It also results in mini facelift (Fig. 14.10).

- If whole of vermillion border of lower lip shows premalignant changes due to UV rays, a total **lip shave** should be included with resection of primary tumor. To cover the defect, mucosa of the inner aspect of the lip is undermined and advanced up to the cutaneous edge (Fig. 14.11).

Carcinoma Tonsil

It is primarily treated with radiotherapy. If there is any residual or recurrent tumor after radiotherapy, it is treated with radical surgery and reconstruction.

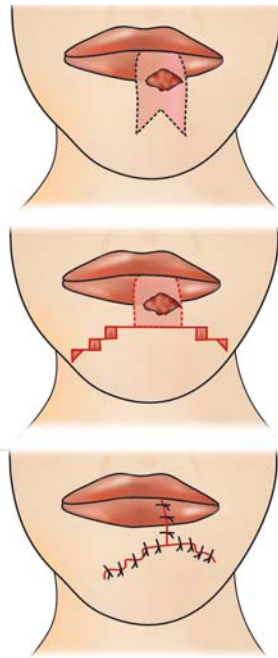


Fig. 14.10: Johansen stepladder procedure

(ii) RADIOTHERAPY

- Radiotherapy and surgery have equal results in controlling early lesions of oral cavity.
- Radiotherapy can be given as external beam or brachytherapy or combination of both.
- Indication and contraindications of radiotherapy are given in Box 14.10.

Box 14.10: Radiotherapy in oral cancers

Indications

- Early oral cancers.
- Patient unfit for surgery.
- Patient unwilling for surgery.
- Down staging of advanced lesions.
- Postoperative radiotherapy for residual lesion.
- Palliative radiotherapy in inoperable lesion

Contraindications

- Gingivoalveolar cancers (risk of radionecrosis)
- Syphilitic glossitis

- The main advantage of radiotherapy is organ preservation.

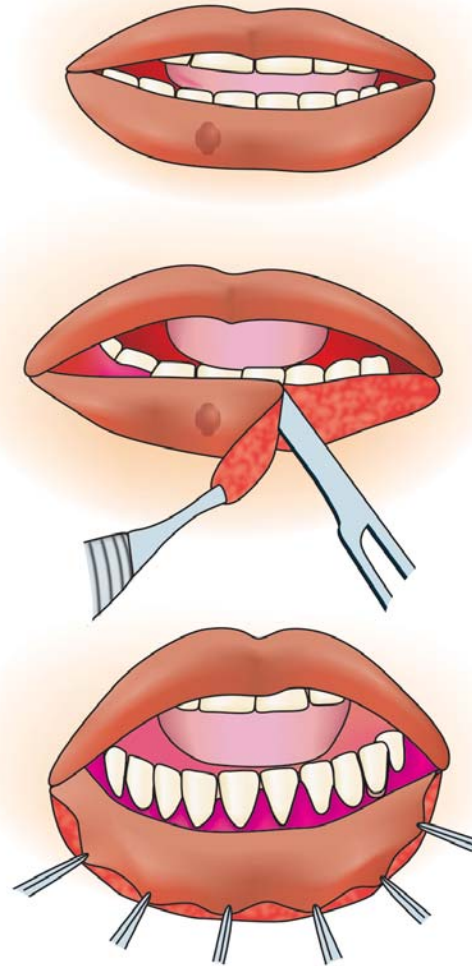


Fig. 14.11: Lip shave procedure

- The total dose is 65-75 Gy to the primary and neck for clinically evident disease.
- The side effects of radiotherapy are given in Box 14.11.

Box 14.11: Side effects of radiotherapy

- Erythema
- Ulceration
- Tissue edema
- Skin sloughing
- Xerostomia
- Dental caries
- Osteoradionecrosis

(iii) CHEMOTHERAPY

- It is mostly used as palliation for advanced or recurrent oral cancers.
- Neoadjuvant chemotherapy (before surgery) can be given to down stage the tumor so that large tumor becomes operable.
- Adjuvant chemotherapy (after surgery) can be given to improve survival.
- Chemotherapy is very effective in verrucous carcinoma.
- Drugs most commonly used are cisplatin, 5 FU, methotrexate, bleomycin and ifosfamide. These are used alone or in combination. Cisplatin based chemotherapy is more effective than single agent chemotherapy.
- Palliative chemotherapy should not be given to the patients with poor performance status because of high-risk of complications.

B. TREATMENT OF NECK NODES

- Treatment of the neck depends upon the status of cervical lymph nodes.

i. N_0 Nodes

- In clinically impalpable nodes, the management options are:

Observation alone

or

Elective block dissection

The recent reports are in favor of **elective block dissection (prophylactic neck dissection)** due to following reasons:

- ☐ In clinically negative neck, about 25-50% nodes are found to be involved with metastatic deposits on histological examination. Thus, elective block dissection also acts as a staging procedure.
 - ☐ Patient may not come for regular follow-up and might come with extensive nodal metastasis.
 - ☐ Once nodal metastasis develops, survival rate is considerably decreased.
 - ☐ Elective block dissection carries negligible mortality and acceptable morbidity.
- In N_0 nodes, supraomohyoid neck dissection is found to be sufficient since it removes majority of histologically positive nodes.

- In N_0 nodes, another good alternative to surgery is radiotherapy of neck nodes. 40 Gy dosage of radiations carry less morbidity than surgery.

ii. Ipsilateral Neck Nodes (N_1 , N_{2a} , N_{2b})

- Treatment of choice is radical neck dissection.
- If lymph nodes are multiple or there is extracapsular tumor spread, postoperative radiotherapy should be given to the neck.
- If patient is not fit for surgery, only radiotherapy should be given.

iii. Bilateral Neck Nodes (N_{2c} , N_3)

- Nodal spread can occur to both sides of the neck especially if the lesion is close to midline.
- The treatment is bilateral neck dissection with preservation of internal jugular vein on at least one side (the less affected side).
- Resection of both internal jugular veins should not be done because it leads to cerebral venous engorgement and high chances of mortality.
- Bilateral neck dissection is usually followed by post-operative radiotherapy because mostly multiple nodes are involved or there is extracapsular spread.
- If primary tumor is large and inoperable along with bilateral neck nodes, treatment is radiotherapy alone for both primary tumor as well as neck nodes.
- If primary tumor and neck nodes become operable after radiotherapy in a young and fit patient, then surgery should be done.

After management of the neck, patient is kept on regular follow-up. If some nodes appear in the neck, FNAC is the investigation of choice that helps in differentiating carcinoma from reactive lymphadenitis. If nodes are positive for metastasis, treatment is radical neck dissection.

Prognosis

- Tongue cancer has poorer prognosis in comparison to other sites in oral cavity.
- Poor prognosis is seen in:
 - ☐ Lymph node metastasis.
 - ☐ Multiple lymph node involvement.
 - ☐ Extracapsular spread.
- Five year survival in early stages (I and II) is 30-100% and in advanced stages (III and IV) is 7-41%.

CLINICAL EXAMINATION OF ORAL CAVITY

History

- Ask about complaints of pain, swelling or ulcer in the oral cavity.
- History of foul smelling breath, hypersalivation, difficulty in speaking, hoarseness of voice and dysphagia are suggestive of oral malignancy.
- Enquire about history of excessive smoking, tobacco chewing and drinking alcohol in personal history.
- In past history, ask about history of tuberculosis or syphilis.

Examination

- It is best done with patient sitting on a stool and examiner standing in front of him.
- The examiner should wear gloves and use torch and tongue depressor for good illumination and exposure (Fig. 14.12).
- General appearance: The patient of oral malignancy appears debilitated with foul smelling breath and drooling saliva at angle of mouth. He cannot articulate clear words.

Inspection

- Look at face for any:
 - ☐ Lesion of the lips.
 - ☐ Abnormality of face, nose eyes, ears.
 - ☐ Orocutaneous fistula.
 - ☐ Multiple neck swellings (lymph nodes).



Fig.14.12: Equipments for oral cavity examination—gloves, torch and tongue depressor



Fig. 14.13: Inspection of tongue

- Ask the patient to open mouth and look for any difficulty in opening mouth (trismus).
- Examine inside of oral cavity using good illumination with a torch.
- Ask the patient to protrude the tongue (Fig. 14.13). Examine for any restricted mobility (ankyloglossia) (Box 14.12) or lateral deviation of tongue (hypoglossal nerve palsy).

Box 14.12: Ankyloglossia

- Carcinoma tongue
- Tongue tie (short frenum linguae)

- Examine the tongue for its:
 - ☐ Size (micro/macroglossia).
 - ☐ Color.
 - ☐ Crack, fissure or ulcer.
 - ☐ Swelling.
- Ask the patient to roll the tongue upwards and examine undersurface of tongue and floor of mouth by bending the head slightly backwards (Fig. 14.14A). In tongue tie, the tongue is unable to touch the palate because lingual frenum is short and thick (Fig. 14.14B).
- Examine teeth for their color, any tar-tar deposition, caries, unerupted tooth, dental sepsis.
- Examine gums for gingivitis, epulis, (pedunculated mass) or any growth.
- Retract angle of the mouth and examine mucosa of the cheek (Fig. 14.15). Look for any pigmented



Figs 14.14A and B: (A) Inspection of floor of the mouth (normal), and (B) Tongue unable to touch the palate (Tongue tie)



Fig. 14.15: Inspection of cheek mucosa



Fig. 14.16: Inspection of palate, tonsils and posterior pharyngeal wall

patch, leukoplakia, retention cyst or growth. Examine the opening of Stenson's duct that lies opposite the upper second molar tooth.

- Ask patient to look up and press the tongue down with a tongue depressor. Examine palate for any cleft, perforation, ulcer, swelling or growth.
- Examine position of uvula and ask patient to say 'Aah...'. In vagus nerve palsy, the affected half of soft palate remains immobile.
- Examine tonsils, pillars of the fauces and posterior pharyngeal wall for any pathology (Fig. 14.16).

Palpation

- Make the patient comfortable by explaining the procedure you are going to do because gag reflex is induced by putting finger in the oral cavity.
- Put gloved finger in the oral cavity and feel for any induration, ulcer, swelling, or growth of the tongue. The induration is assessed by asking the patient to keep the tongue relaxed within the oral cavity. It is because on protrusion, the contracted muscles of tongue give false impression of induration (Figs 14.17A and B).
- Always palpate posterior 1/3rd of tongue for any ulcer, induration or growth. During examination, the



Figs 14.17A and B: (A) Palpation for induration while tongue is inside the oral cavity (correct method), and (B) Palpation for induration while tongue is protruded (incorrect method)



Fig. 14.18: Palpation of posterior third of tongue. Left index finger is pushed in between two jaws to keep the mouth open



Fig. 14.19: Bimanual palpation of cheek

examiner pushes his left index finger in between upper and lower jaws of the patient to prevent closing of mouth and biting of the fingers (Fig. 14.18).

- Palpate teeth for any tenderness, loose teeth or any other abnormality.
- Palpate gums for any tenderness, ulcer or mass. Pus might exude on pressing gums in case of dental sepsis.
- Palpate floor of the mouth bimanually to feel for any pathology. Enlarged submandibular gland is bimanually palpated and a stone may be palpable in the Wharton's duct (Fig. 15.19). In case of a cystic swelling in floor of mouth, do transillumination test.

A ranula is transilluminant while sublingual dermoid is not.

- Palpate cheek bimanually with a finger in mouth and thumb outside so that its mucosal as well as skin surfaces are examined for any lesion (Fig. 14.19).
- Palpate palate, tonsils and posterior pharyngeal wall with right index finger while the left index finger keeps the jaws open as described above. Before palpation, these areas should be sprayed with local anesthetic agent (xylocaine spray) to prevent gag reflex.
- Palpate all cervical lymph nodes for any enlargement.

15

Chapter

Diseases of Salivary Glands

Sanjay Marwah

Salivary glands are divided into two groups as **major** and **minor** glands.

There are three pairs of **major** salivary glands

- Parotid
- Submandibular
- Sublingual

Hundreds of **minor** salivary glands lie in submucosa of upper aerodigestive tract like lips, cheek, floor of mouth, oropharynx, trachea, larynx and palate. Overall they contribute to 10% of salivary volume. The **function** of salivary glands is to keep the oral cavity moist and lubricated. The salivary volume secreted by the glands is 1500 ml/day.

Histologically, salivary glands are:

- Serous: Parotid glands
- Mucus: Sublingual and minor glands
- Mixed: Submandibular glands

Embryologically, the glands are formed by ectodermal and endodermal invagination.

SURGICAL ANATOMY

Parotid Gland

- It is located in the retromandibular fossa in an area anterior and inferior to external auditory canal (Fig. 15.1).
- The duct of the gland (Stensen's duct) opens in the mucosa of cheek opposite to upper second molar tooth.
- There are three important nerves in relation to parotid gland (Box 15.1).
- The extracranial part of facial nerve divides the gland into **superficial** (80%) and **deep** (20%) parts.
- Facial nerve alongwith retromandibular vein makes a plane named 'faciovenous plane of Patey' in the substance of parotid gland.
- Greater auricular nerve enters tail of parotid gland and it is sensory to tragus area and ear lobe.
- Auriculotemporal nerve is branch of mandibular division of 5th cranial nerve. It contains parasympathetic fibers sent to parotid by otic ganglion.

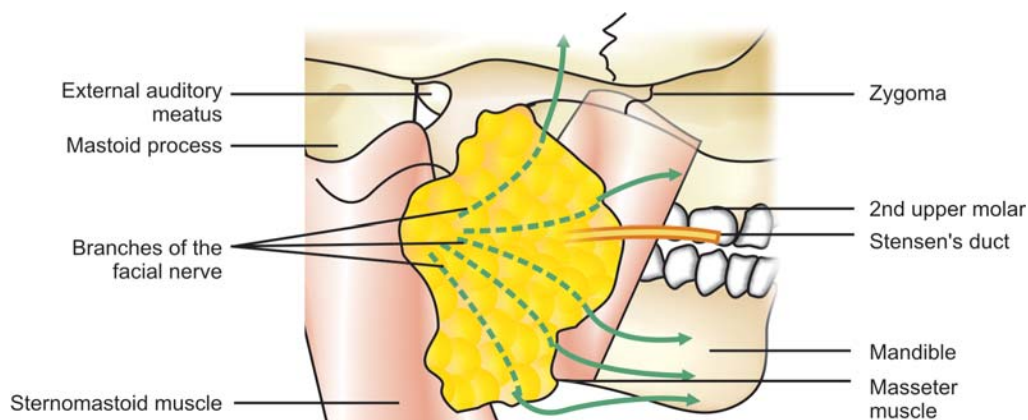


Fig. 15.1: Surgical anatomy of parotid gland

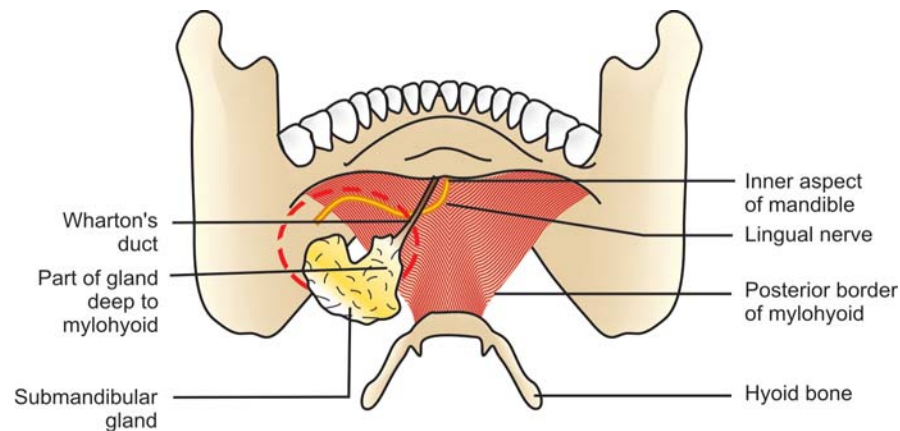


Fig. 15.2: Surgical anatomy of submandibular gland; floor of the mouth viewed posteriorly after tongue removal

- Facial nerve emerges through stylomastoid foramen. The anatomical landmark for its location is the point where tip of mastoid, cartilaginous auditory canal and posterior belly of digastric muscle meet. The nerve enters the substance of parotid gland and divides into two main branches—upper temporo-facial and lower cervicofacial divisions. The upper division further divides into zygomatic, temporal and buccal branches while the lower division divides into mandibular and cervical branches (Box 15.1A).

Submandibular Gland

- The gland is located in the digastric triangle.
- The gland has **superficial** and **deep** parts.
- The **superficial** part is located in submandibular space within digastric triangle overlying mylohyoid and hyoglossus muscles.
- The **deep** part is deep to mylohyoid muscle.
- The two parts join round the posterior free edge of mylohyoid muscle (Fig. 15.2B).
- There are three important nerves in relation to the gland (Box 15.1B).

Box 15.1A: Distribution of facial nerve branches

Facial nerve branch	Muscles supplied
Temporal	Frontalis
Zygomatic	Orbicularis oculi
Buccal	Orbicularis oris, Buccinator
Mandibular	Lower lip muscles
Cervical	Platysma

Box 15.1B: Three important nerves in relation

Parotid gland

Facial nerve
Greater auricular nerve
Auriculotemporal nerve

Submandibular gland

Mandibular nerve (Branch of facial nerve)
Sublingual nerve
Hypoglossal nerve

- The facial artery enters the posterior aspect of deep surface and deeply grooves the gland.
- The duct (Wharton's duct) runs from the deep lobe and opens on floor of the mouth lateral to frenulum of the tongue.

Sublingual Gland

- It is located just beneath mucosa of floor of mouth.
- It drains by several small ducts directly into oral cavity or into submandibular duct.

DISEASES OF SALIVARY GLANDS

Mucous Retention Cyst

It is one of the most common salivary gland disorders. It usually develops from obstructed minor salivary gland. It produces a painless and translucent swelling in oral mucosa mostly in the lower lip or cheek (Fig. 15.3). Smaller cysts may resolve spontaneously but larger cysts require surgical excision.



Fig. 15.3: Mucus retention cyst lower lip

Ranula

It is mucus extravasation cyst originating in sublingual salivary gland. It produces a translucent cystic swelling with a bluish tinge situated on floor of mouth on one side of fraenum linguae. These findings are characteristically described as “frog’s belly appearance”.

Sometimes mucus extravasation involves both sublingual and submandibular glands. So the ranula extends into the neck and becomes bimanually palpable. It is called a *Plunging ranula* (Fig. 15.4).

The ranula needs to be differentiated from *sublingual dermoid cyst* that also presents as a cystic swelling in the floor of mouth. It contains sebaceous material and is opaque that completely differs from brilliant translucency of a ranula.

The *diagnosis* of ranula is essentially clinical. However, ultrasound and MRI scan can help in outlining the extent of lesion.



Fig. 15.4: Plunging ranula seen in floor of mouth as well as submandibular region

Treatment is surgical excision of the cyst along with affected sublingual gland through transoral route. In case of plunging ranula, excision of the cyst along with both sublingual and submandibular gland is done through cervical incision.

Simple incision and drainage of the cyst leads to recurrence.

INFLAMMATORY DISORDERS

Submandibular Sialadenitis

It is mostly chronic and rarely acute in nature. *Acute sialadenitis* may occur due to viral (mumps) or bacterial infection. But most of the times, it becomes chronic infection because of poor healing capacity of the submandibular gland.

Chronic sialadenitis mostly occurs due to stone formation in submandibular gland or its duct system. 80% of salivary stones occur in submandibular gland because:

- Secretion is thick, mucus in nature.
- Antigravity drainage of secretion since duct opens at a higher level than the gland.
- The Wharton’s duct is kinked by lingual nerve (see Fig. 15.2).

Clinical Features

Most of the times, stone produces partial obstruction of the duct and patient presents with mildly painful submandibular swelling (Fig. 15.5).

In case of complete obstruction, patient presents with painful swelling in submandibular region appearing during



Fig. 15.5: Left submandibular gland enlargement due to chronic sialadenitis; swelling was palpable bimanually

meals and disappearing 1-2 hours after meals. On examination, submandibular gland is tender, firm and palpable bimanually. (cf submandibular lymph node: not palpable bimanually). Sometimes stone may be palpable in the gland or in the duct (in floor of mouth). The opening of the duct at sublingual papilla may exude pus.

Diagnosis

Since 80% stones are radiopaque, an oblique lateral or posterior oblique occlusal X- ray may show stone in the submandibular region.

Differential Diagnosis

See Box 15.2.

Box 15.2: Swelling submandibular region

Causes

- Submandibular sialadenitis
- Submandibular tumor
- Submandibular lymphadenopathy
- Plunging ranula
- Cystic hygroma

Treatment

The stone lying in the duct can be removed by incising the duct over stone in floor of mouth. After stone removal, the opening in the duct should be left unsutured for free drainage of saliva.

If stone is palpable in proximal duct (lateral to second molar region) it should not be removed through intra-oral route since there is risk of damage to lingual nerve that hooks the duct in this region. In such cases or in cases having stone in the gland, treatment is submandibular gland excision.

Submandibular gland excision:

- Incision is given 3-4 cm. below and parallel to margin of the mandible to avoid damage to marginal mandibular branch of facial nerve.
- Skin, platysma and deep fascia are incised to expose the superficial part of the gland.
- Gland is mobilized and facial artery ligated and divided at posterior pole of the gland in a deeper plane.
- The facial artery needs to be ligated again at lower border of mandible in a superficial plane.
- Posterior border of mylohyoid muscle is exposed and retracted forwards.
- Deep lobe (deep to mylohyoid muscle) is mobilized and retracted down to expose the Wharton's duct.

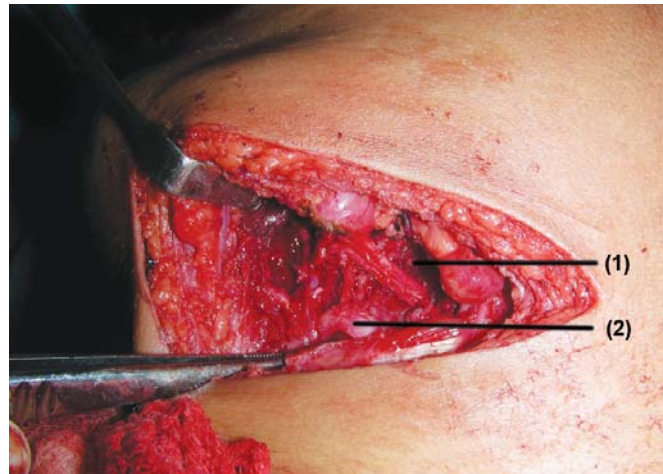


Fig. 15.6: Lingual (1) and hypoglossal nerves (2) exposed after excision of submandibular gland

The lingual nerve is attached to the deep lobe through parasympathetic fibers that are divided to free the lingual nerve.

- The duct is divided taking care not to injure the lingual nerve (Fig. 15.6).
- Wound is closed over a closed suction drain.

Complications

- Bleeding and hematoma formation.
- Infection.
- Injury to marginal mandibular nerve (drooping of angle of mouth).
- Injury to lingual nerve (anesthesia of anterior 2/3rd of tongue).
- Injury to hypoglossal nerve (unilateral tongue paralysis).

Acute Parotitis

Acute painful swelling of parotid gland due to inflammation (Fig. 15.7). Various causes are:

Viral Infection

- Mumps is the most common cause of acute parotitis.
- Maximum incidence is in children (4-6 years of age).
- Usually there is history of contact with infected child.
- Initial symptoms are fever, malaise and headache followed by painful swelling of one or both parotid glands.
- Treatment is symptomatic in form of antipyretics and oral fluid.



Fig. 15.7: Acute parotitis

- Complications are serious although uncommon and mostly seen in adults. These are pancreatitis, meningitis, orchitis and sudden deafness.

Bacterial Infection

- Ascending bacterial infection from oral cavity leads to acute suppurative parotitis.
- It is usually seen in postoperative period when patient is dehydrated and has poor oral hygiene.
- Calculi and duct stricture are other predisposing factors.
- The infecting organism is usually *Staph aureus*.
- Patient presents with fever and diffuse painful enlargement of the gland.
- Purulent saliva can be seen at the duct orifice especially on gentle massage of the gland.
- Initial treatment is conservative in form of hydration, oral hygiene and parenteral antibiotics
- If patient does not improve and the swelling increases, it is suggestive of parotid abscess.
- The abscess remains nonfluctuant due to overlying parotid fascia.
- The abscess can be located with USG or CT scan.
- Treatment is evacuation of pus either by wide bore needle aspiration or surgical drainage under general anesthesia.
- In surgical drainage, skin incision is given low in preauricular region to avoid damage to facial nerve branches.
- Blunt dissection with sinus forceps is done to break the loculi (Hilton's method).

Obstructive Causes

a. Papillary obstruction

- Ill-fitting denture causing trauma, inflammation and blockage of parotid duct papilla.
- The patient presents with painful swelling of parotid gland during meal times.
- Treatment is papillotomy that allows free drainage of saliva.

b. Parotid calculi

- Stones in parotid gland are less common in comparison to submandibular gland stones.
- Stones are usually radiolucent and are identified on sialography.
- Clinical presentation is of painful swelling during meals.
- Treatment is surgical removal of the stone.

Granulomatous Sialadenitis

It is a rare group of conditions that include:

Tuberculosis

The organism usually gains access to the salivary glands by a lesion in oral cavity, teeth or tonsil and presents as a painless mass in the gland. FNAC of the mass helps in reaching the diagnosis and patients mostly respond to antitubercular treatment. Sometimes diagnosis is established after surgical excision of the gland.

Actinomyces

Extension into the salivary glands occurs from adjacent sites causing firm, indurated mass. It leads to fistula formation producing yellow 'sulphur' granules. Treatment is with extended course of penicillin.

Sarcoidosis

It mostly affects the parotid gland producing localized tumor like swelling (sarcoid pseudotumor). **Heerfordt syndrome** (uveoparotid fever) is a form of sarcoidosis characterized by fever, parotid swelling, uveitis and facial palsy. The diagnosis is confirmed by biopsy revealing noncaseating granulomas. Treatment is with steroids.

Cat-scratch Disease

It usually affects children and caused by *Bartonella henselae*. A pustule forms at the site of infection following a scratch by a cat. It is followed by cervical

lymphadenopathy and parotid gland enlargement. It is a self-limiting condition and resolves without treatment.

Sialadenosis

It is non-inflammatory, non-neoplastic enlargement of salivary glands usually affecting the parotid glands. Various causes are:

- Malnutrition
- Obesity
- Alcoholic cirrhosis
- Pregnancy
- Endocrine disorders (Diabetes, Hypothyroidism, Cushing's disease)
- Drug induced (anticholinergics, sympathomimetics, antidepressants, methyl dopa, hydrochlorothiazide)
- Idiopathic

The treatment is unsatisfactory and usually aims at correcting the underlying cause.

Pseudoparotomegaly

It mimics parotid enlargement and its causes are:

- Hypertrophic masseter
- Winged mandible
- Mandibular tumor
- Preauricular lymph node
- Dental cyst
- Branchial cyst
- Facial nerve neuroma

Various causes of preauricular swelling are given in Box 15.3.

Box 15.3: Preauricular swelling—causes

- Parotid tumor
- Preauricular lymphadenopathy
- Branchial cyst
- Lateral dermoid cyst
- Cystic hygroma

SALIVARY GLAND TUMORS

Epidemiology

- They comprise 3% of all head and neck tumors.
- Majority of tumors occur in major salivary glands most commonly in parotid gland.
- Majority of tumors are seen in adults.
- In young children, parotid swelling is most likely to be a hemangioma or lymphangioma.
- Majority of tumors occurring in parotid glands are benign while majority in minor glands are malignant.

- In submandibular gland, benign and malignant tumors occur with equal frequency while in sublingual gland, tumors are almost always malignant (Box 15.4).

Box 15.4: Percentage risk of benign vs malignant tumors in salivary glands

	<i>Benign</i>	<i>Malignant</i>
Parotid gland	75%	25%
Submandibular gland	50%	50%
Sublingual gland	05%	95%
Minor salivary glands	25%	75%

Etiology

As for most of the other cancers, exact etiology of salivary gland tumors is unknown. However, some environmental factors may increase the risk of tumors in salivary glands. These are:

- Low dose ionizing radiation to head and neck (in dental X-rays). High dose radiotherapy has no role.
- EB virus.
- Occupational hazard: Exposure to hard wood dust, nitrosamines (rubber industry).
- Tobacco: Warthin's tumor is strongly associated with smoking.
- Use of cellular telephone.

Histological Classification

A. Epithelial Tumors

Adenoma

Pleomorphic adenoma
Monomorphic adenoma
(Warthin's tumor)

Carcinoma

Low grade

Acinic cell carcinoma
Adenoid cystic carcinoma
Low grade mucoepidermoid carcinoma

High grade

Squamous cell carcinoma
Adenocarcinoma
Carcinoma in pleomorphic adenoma
High grade mucoepidermoid carcinoma

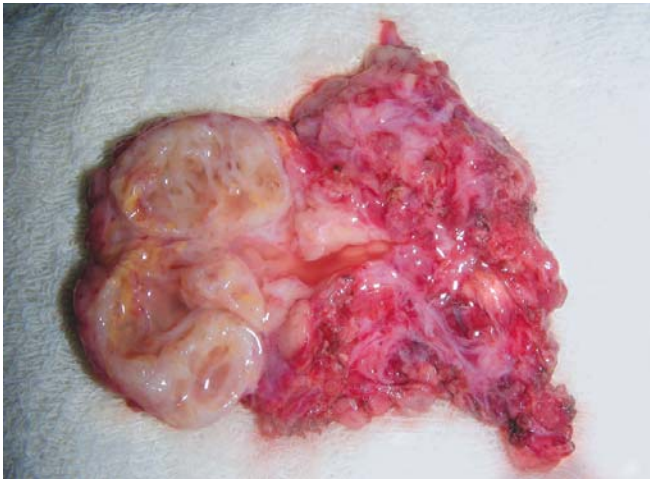


Fig.15.8: Cut section of pleomorphic adenoma in superficial parotidectomy specimen

B. Non-epithelial Tumors

Angioma
Lipoma
Neurofibroma

Others

Lymphoma
Sarcoma
Metastatic tumors

PAROTID GLAND TUMORS

Pleomorphic Adenoma

It is also known as mixed parotid tumor. The characteristic **pathological features** (Fig. 15.8) are:

- Epithelial and myoepithelial cells proliferate in sheets and strands.
- Pseudocapsule (formed by compressed parotid tissue around the tumor).
- Pseudocartilag (mucoid material separating epithelial cells give appearance of cartilage).
- Pseudopodia (strands of tumor cells project through the pseudocapsule into adjoining part of the parotid gland).

Due to presence of pseudopodia, simple enucleation of tumor will leave behind residual tumor leading to recurrence. Hence, superficial parotidectomy is the recommended treatment even for a benign tumor.

Clinical Features

- Most tumors are located in superficial lobe.



Fig. 15.9: Left pleomorphic adenoma raising ear lobule and obliterating retromandibular area

- Clinically, it presents as a painless slow growing mass that is rubbery and nodular in consistency.
- The mass classically raises the ear lobule and obliterates the retromandibular groove (Fig. 15.9).
- Even in big tumor, the mass is not adherent to overlying skin or underlying masseter muscle and there is no facial palsy.
- Deep lobe tumors present with fullness in retromandibular area along with soft palate swelling.
- Long standing pleomorphic adenoma may convert to carcinoma in 5% cases.
- The signs of malignant change are:
 - ☐ Sudden increase in tumor size.
 - ☐ Facial nerve palsy (Fig. 15.10).
 - ☐ Tumor consistency becomes hard.

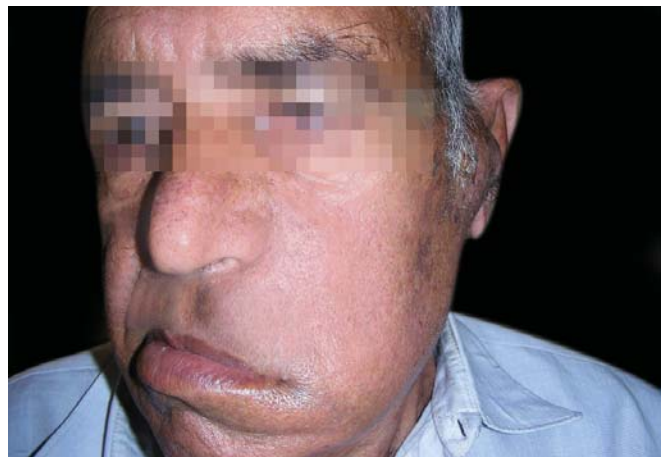


Fig. 15.10: Left facial nerve palsy due to malignant parotid tumor



Fig. 15.11: Left malignant parotid tumor with cervical lymph node metastasis

- ☐ Tumor becomes fixed to underlying structures and invades overlying skin.
- ☐ Cervical lymph node enlargement (Fig. 15.11).
- ☐ Limited jaw movements due to mandibular invasion.

Warthin's Tumor

It is a benign tumor occurring next to pleomorphic adenoma. It forms 10% of parotid tumors. It is also called as adenolymphoma. It is not a true lymphoma but this name is given due to presence of lymphoid tissue in the tumor. It consists of cystic spaces lined by double layered epithelium.

It usually affects middle aged or elderly males. It presents as a slow growing soft swelling at lower pole of parotid gland (Fig. 15.12).



Fig. 15.12: Soft slow growing parotid swelling in elderly male—Warthin's tumor

Box 15.5: Pleomorphic adenoma vs Warthin's tumor

	<i>Pleomorphic adenoma</i>	<i>Warthin's tumor</i>
Incidence	Most common (80%)	Less common (10%)
Age and sex	Middle aged females	Elderly males
Occurrence	Unilateral	Sometimes bilateral
On examination	Firm, nodular	Cystic, smooth
Histopathology	Sheets and strands of epithelial and myoepithelial cells	Cystic spaces and lymphoid tissue
Tc ^{99m} scan	Cold spot	Hot spot
Treatment	Superficial parotidectomy	Enucleation

It produces a hot spot on ^{99m}Tc isotope scan.

For treatment, simple enucleation can be done since its capsule is well-defined.

Box 15.5 shows comparison of Warthin's tumor with pleomorphic adenoma.

Acinic Cell Tumor

It is almost always seen in parotid gland. It is a low grade tumor and is composed of serous acini. It is soft and cystic. Rarely it may metastasize.

Mucoepidermoid Tumor

It is composed of sheets and masses of epidermoid cells and cystic spaces lined by mucus secreting cells. Low grade tumor has more cystic spaces and fewer cells while high grade tumor has more cells and less cystic spaces. It is slow growing in case of low grade tumor and rapidly growing with metastasis to lymph nodes and lungs in high grade tumor (Fig. 15.13). It is most common salivary neoplasm in children. Clinically, patients present with a hard mass in the gland.

Adenoid Cystic Carcinoma

It is most common malignant salivary tumor. It consists of myoepithelial and duct cells, which are arranged in sheets and cords and give *cribriform or lace like appearance*. Its characteristic feature is **perineural invasion** and **bone invasion**. Thus, tumor is always more extensive than seen clinically or on radiograph. Clinically, it presents as a hard fixed mass. Later, patient



Fig. 15.13: Rapidly growing and fungating parotid tumor in a child—high grade mucoepidermoid tumor

may complain of pain, anesthesia of overlying skin and muscle paralysis due to involvement of related nerves.

SUBMANDIBULAR GLAND TUMORS

They are uncommon and usually present as a painless slow growing swelling in submandibular triangle (Fig. 15.14). In 50% cases, they are benign in nature. The submandibular gland tumor is bimanually palpable. This clinical sign helps in differentiating it from submandibular lymphadenopathy which is not palpable bimanually. Treatment is submandibular gland excision with a cuff of normal tissue.

MINOR SALIVARY GLAND TUMORS

- They arise from mucous glands of upper aerodigestive tract and majority is malignant.
- Commonest site is on hard palate followed by lip and retromolar region.



Fig. 15.14: Tumor left submandibular gland

- Commonest type is adenoid cystic carcinoma.
- It usually presents as a firm mass which later undergoes necrosis and ulceration. It may invade adjoining soft tissues, bone and nerve.
- Treatment is wide excision with plastic reconstruction.

Investigations for Salivary Gland Tumors

Radiological Evaluation

- Diagnostic imaging is not required routinely.
- *Plain X-ray, USG and sialography* have no definite role in salivary tumors.
- *CT scan* and *MRI* are good for evaluation of malignant masses that are deep seated and fixed.
- CT scan and MRI help in defining location and extent of tumor, evaluation of neck nodes.
- Bone destruction is best seen on CT scan.
- MRI is useful in detecting perineural invasion, intracranial extension of tumor and detecting deep lobe parotid tumors.
- *PET scan* is superior to CT and MRI in detecting local recurrence and distinguishing it from past treatment fibrosis.

Cytopathological Diagnosis

- Preoperative tissue diagnosis is not required in discreet parotid swelling.
- *FNAC* is done when there is high clinical suspicion of malignancy.
 - ☐ Surgery is modified if report is lymphoma. In such case, only incision biopsy is done instead of tumor excision so as to grade the tumor.
 - ☐ If report is Warthin's tumor in an old patient, the treatment is conservative (no surgery).
 - ☐ FNAC is also useful in recurrent and inoperable tumors for planning radiotherapy as initial treatment.
- *Open biopsy* is not done routinely due to risk of injury to facial nerve and spreading of tumor cells. Biopsy is indicated in following conditions:
 - ☐ Repeated FNAC inconclusive in a hard fixed mass.
 - ☐ In case of lymphoma for tumor grading.

Treatment of Salivary Gland Tumors

- *Benign* and slow growing neoplasm confined to superficial lobe of parotid gland is treated with superficial parotidectomy with facial nerve conservation. In deep lobe tumors, first superficial parotidectomy

with identification of facial nerve is completed. Then with blunt dissection, deep lobe tumor is removed from in-between nerve branches.

- **Malignant** and high grade parotid tumors require superficial/total/radical parotidectomy with or without sacrifice of facial nerve depending on tumor extent.
 - Radical parotidectomy may include removal of whole parotid gland with facial nerve, adjoining muscles (masseter, pterygoids) and mandible.
 - Most important aim of surgery in malignant tumor is "To achieve clear margins of resection". Clearance of surgical margins can be confirmed by intraoperative frozen section of the excised specimen (Box 15.6).

Box 15.6: Frozen section evaluation

- Confirms neoplasm (benign vs malignant)
 - Accuracy 80-90%
 - Confirms margin clearance
 - Lymph node assessment for metastasis
- If biopsy of resected specimen even in radical parotidectomy shows positive margins for tumor, there is high-risk of recurrence and decreased survival (Figs 15.15A and B).
 - On the other hand, if surgical margins are negative even in superficial parotidectomy, it is adequate.
 - Thus **more** surgery does not improve survival.

Box 15.7: Superficial parotidectomy

- Treatment for pleomorphic adenoma
- Superficial lobe along with tumor is removed
- Facial nerve branches are identified and preserved
- Avoids tumor spillage and removes pseudopodia
- Simple enucleation will leave behind residual tumor leading to recurrence

Superficial Parotidectomy (Box 15.7)

Important steps of surgery are:

- 'Lazy S' incision is given which extends from preauricular to mastoid and then in cervical region.
- Skin flaps are raised to expose parotid gland anteriorly and sternomastoid and posterior belly of digastric muscle posteriorly.
- An avascular plane is developed in preauricular area anterior to mastoid tip requiring division of greater auricular nerve.
- By further dissection, facial nerve trunk is identified with the help of various anatomical landmarks (Box 15.8).

Box 15.8: Anatomical landmarks for facial nerve identification

- Tragal pointer (Tragal cartilage points towards nerve)
 - Nerve lies deep and medial to tip of mastoid process
 - Posterior belly of digastric muscle (lies just inferior and parallel to nerve)
 - Styloid process (lies medial and anterior to nerve)
- Bipolar cautery is used for hemostasis to prevent facial nerve damage.



Figs 15.15A and B: Recurrent malignant parotid tumor after superficial parotidectomy (lateral and frontal views); previous biopsy report revealed positive margins for tumor



Fig. 15.16: Facial nerve branches exposed following superficial parotidectomy

- After identification of facial nerve trunk, its branches are dissected towards periphery by dissecting in perineural plane (Fig. 15.16).
- Facial nerve can be traced retrograde as well by identifying one of its branches at periphery.
- The superficial lobe along with tumor is removed *in toto*.
- The wound is closed over a negative suction drain.

Facial Nerve Management

- Preoperative functional status of facial nerve should be assessed by physical examination and look for any partial or total facial nerve palsy.
- In case of absence of clinical nerve involvement and presence of surgical plane between tumor and the nerve during operation, the facial nerve should be preserved.
- In case of preoperative facial palsy and operative findings of nerve invasion by the tumor, the involved area of nerve should be resected.
- After nerve resection, immediate reconstruction by sural or greater auricular nerve should be done and its success rate is 75%.
- Rehabilitation procedures should be performed simultaneously in form of:
 - ☐ Gold weight upper eyelid implants.
 - ☐ Lower lid tightening.
 - ☐ Static facial slings.
- Details of management of facial nerve palsy are given in Chapter 17: Head Injury.

Management of Neck Nodes

- Neck dissection should be done in node positive cases only.
- Modified radical neck dissection is the preferred technique.
- In high grade tumors with clinically negative neck nodes, frozen section of suspicious nodes should be done.
- There is no benefit of elective node dissection in clinically negative neck.

Role of Radiotherapy

- It is always indicated in high grade malignant tumors for improving local control.
- Area of radiotherapy includes preoperative extent with 2 cm margin.
- In adenoid cystic carcinoma, radiotherapy is also given to named nerve roots up to the base of skull.
- In inoperable tumors, radiotherapy is given for palliation.
- In case of recurrent malignant tumors, if resection is not possible, then high dose radiotherapy is given as:
 - External beam RT,
 - Neutron RT or
 - Brachytherapy

RT in Pleomorphic Adenoma: Indications

- Deep lobe tumors
- Recurrence after surgery
- Microscopically positive margins
- Significant tumor spillage

Chemotherapy has no proven role.

Management protocol of salivary gland tumors is given in Box 15.9.

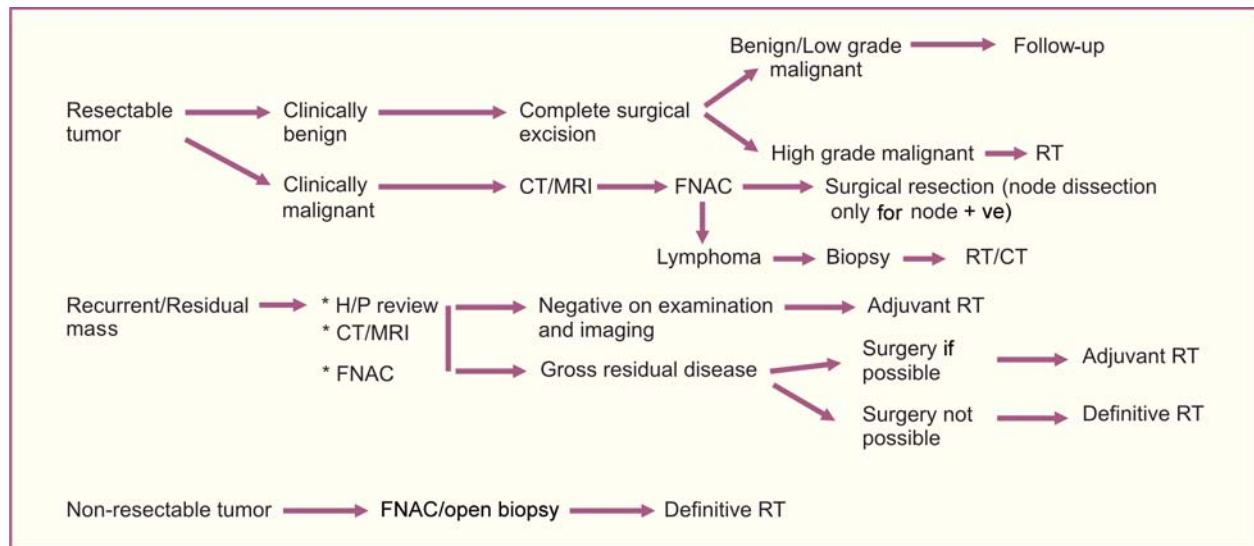
Complications of Parotidectomy

See Box 15.10.

Frey's Syndrome

It is also known as gustatory sweating. It is a relatively common long-term complication after parotidectomy. It results from damage of salivary gland innervation during dissection. There is inappropriate regeneration of parasympathetic fibers, which start stimulating sweat

Box 15.9: Salivary gland tumors—management protocol



Box 15.10: Complications of parotidectomy (5 F)

- Flap necrosis
- Fluid collection (hematoma, infection)
- Facial nerve palsy
- Fistula (salivary fistula)
- Frey's syndrome
- Others—sensory loss to lower pinna (greater auricular nerve damage)

glands of overlying skin. As a result, sweating and skin flush occurs during salivary stimulation.

Most of the patients have mild symptoms and improve after reassurance. Less than 10% cases request for the treatment. Frey's syndrome can be prevented by inserting temporalis fascial flap between skin and parotid bed during parotidectomy.

Treatment is with

- Topical anticholinergic agent (1% Glycopyrrolate).
- Botulinum toxin injection into affected skin.
- Denervation by division of lesser superficial petrosal nerve.

Sjögren's Syndrome

- It is an autoimmune syndrome causing progressive destruction of salivary and lacrimal glands.
- *Primary Sjögren's syndrome* is characterized by presence of dry eyes (keratoconjunctivitis sicca) and dry mouth (xerostomia) (Box 15.11).

- If these features are associated with some connective tissue disorder, it is called as *secondary Sjögren's syndrome*.
- Females are affected ten times more than males.
- There is painful enlargement of salivary glands.
- Sialography shows punctate sialectasis.
- Biopsy of minor salivary glands show focal lymphocytic infiltration.
- *Treatment* is symptomatic in form of artificial tears for dry eyes and oral hydration along with salivary substitutes for dry mouth.
- There is increased risk of developing lymphoma especially in primary Sjögren's syndrome.

Box 15.11: Xerostomia (dry mouth): Causes

- Dehydration
- Anxiety
- Drugs (anticholinergic)
- Sjögren's syndrome
- Post RT in head and neck

CLINICAL EXAMINATION OF SALIVARY GLANDS**History**

- Swelling
 - Most cases present with swelling of the affected gland.
 - Ask for duration and progress of the swelling.

- iii. Commonest cause of parotid gland swelling is pleomorphic adenoma. It is slow growing and may be present for several years. Sudden increase in size of swelling is suggestive of malignant transformation.
- iv. Commonest cause of submandibular gland swelling is chronic sialadenitis due to stone disease. The swelling is of long duration and increases in size during meals.
- b. Pain
 - i. In acute parotitis there is severe throbbing pain.
 - ii. In malignant parotid tumor there can be pain in parotid region with radiation to ear lobule due to involvement of greater auricular nerve.
 - iii. In submandibular sialadenitis, the swelling becomes painful during meals.
- c. Fever: High grade fever in acute parotitis, parotid abscess.
- d. Discharge
 - i. Foul smelling purulent (sometime blood stained) discharge in oral cavity is seen in chronic submandibular sialadenitis.
 - ii. Watery discharge over parotid region is seen in parotid fistula. This follows previous surgery or trauma in parotid region (Box 15.12).
- e. Sialorrhea: Increased salivary flow (Box 15.13).

Examination

Parotid Gland

- Parotid swelling is usually present below ear lobule. The ear lobule is raised and retromandibular sulcus (groove between mandible and mastoid process) is obliterated due to the swelling.
- On palpation, look for exact dimensions, surface, consistency, mobility, fixity to overlying skin and underlying structures (Box 15.14).

Box 15.12: Parotid fistula

Etiology	Superficial parotidectomy Drainage of parotid abscess Trauma of parotid region
Clinical features	Clear watery discharge on cheek, more during meals
Diagnosis	Fistulography Sialography
Treatment	Mostly heals with conservative treatment, Excision of fistula tract with ligation of parotid duct

Box 15.13: Sialorrhea (increased salivary flow): Causes

- Stomatitis
 - Drugs
 - Cerebral palsy
 - Cretinism
- If overlying skin can be pinched over the swelling, it means skin is free (Fig. 15.17).



Fig. 15.17: Testing fixity of swelling to overlying skin by pinching

Box 15.14: Clinical findings of parotid gland tumors

	<i>Pleomorphic adenoma</i>	<i>Adenolymphoma</i>	<i>Carcinoma</i>
Surface	Nodular	Smooth	Irregular
Consistency	Variable (firm, nodular)	Cystic	Hard
Mobility	Mobile	Mobile	Fixed
Overlying skin	Free	Free	Fixed
Underlying structures	Free	Free	Fixed
Facial nerve palsy	Absent	Absent	Present
Cervical lymph nodes	Not enlarged	Not enlarged	Enlarged



Fig. 15.18: Testing mobility of swelling by side-to-side movements

- Fixity to underlying masseter muscle – look for the mobility of swelling and then ask the patient to clinch the teeth so as to contract the masseter muscle. If swelling was earlier mobile and becomes fixed on contraction of masseter muscle, it means the swelling is infiltrating the muscle (Fig. 15.18).
- If swelling is immobile on underlying structures even without clenching teeth, it means it is adherent to underlying mandible as well.
- Clinical examination for facial nerve involvement (see Chapter 17: Head Injury).
- Palpate cervical lymph nodes. Hard lymph nodes are suggestive of metastatic deposits from malignant tumor.
- Examine oral cavity—fullness of lateral pharyngeal wall is seen in deep lobe tumors.
- Examine opening of parotid duct in the vestibule against upper second molar tooth. It may be inflamed in acute parotitis.



Fig. 15.19: Bimanual palpation of floor of mouth

Submandibular Gland

- It is felt as a firm, mildly tender swelling in the submandibular triangle of neck in case of chronic sialadenitis.
- Do bimanual palpation of gland by putting one finger in oral cavity to feel floor of the mouth while other finger feels the submandibular gland from outside (Fig. 15.19). The enlarged gland is bimanually palpable while the enlarged submandibular lymph node is palpable only from outside. Sometimes a hard stone may be palpable in the duct of the submandibular gland.
- Look for the opening of the submandibular duct lateral to the frenulum of tongue that might exude pus in chronic sialadenitis.
- Do examination of all salivary glands on both sides since these might be involved due to autoimmune disease (Sjögren's syndrome).

16

Chapter

Diseases of the Larynx

Sanjay Marwah

SURGICAL ANATOMY

The larynx is a complex box made of bone and cartilage and lined with mucosa. It extends from tip of epiglottis to the distal rim of cricoid cartilage. It is divided into three anatomical areas:

- Supraglottic area*: It extends from epiglottis to the ventricle including pre-epiglottic space, hyoid bone, arytenoid processes and false vocal cords.
- Glottic area*: It includes true vocal cords and anterior commissures.
- Subglottic area*: It is the area surrounded by cricoid cartilage.

PHYSIOLOGY

The main function of larynx is modulation of air inspired through the nose and expired from the lungs for maintenance of normal speech. It coordinates respiration with the swallowing so that food is prevented from entering the respiratory tree and air from entering the digestive tract.

STRIDOR

Stridor means noisy breathing. It can be:

- Inspiratory*: It is due to obstruction at or above vocal cords and commonest cause is inhaled foreign body.
- Expiratory*: It is due to lower respiratory tract problems, e.g. asthma, tracheobronchitis.
- Biphasic*: It is due to obstruction or disease of tracheobronchial airway.

In children, often there is history of foreign body ingestion and the child is cyanosed with inspiratory stridor.

The foreign body should be immediately dislodged by hooking with finger or by inverting the child and slapping the back. In adults, Heimlich maneuver is done

for dislodgement of foreign body. In urgent cases, tracheostomy may be required. In less urgent cases, lateral radiograph of the neck and chest X-ray are done followed by direct laryngoscopy under anesthesia.

EPIGLOTTITIS

It is acute edema of aryepiglottic folds and epiglottis.

Causes

- Infection caused by *H. influenzae*, streptococci, diphtheria.
- Ludwig's angina
- Trauma
- Burns and scalds of head and neck region
- Extension of local malignancy
- Radiotherapy

Clinical Features

- Hoarse voice
- Dysphagia
- Dyspnea

Laryngoscopic examination reveals intense inflammation of aryepiglottic folds and epiglottis.

Treatment

- Steam inhalation
- Local spray of dilute adrenaline solution.
- Antihistaminics and steroids.
- Antibiotics (ampicillin or chloramphenicol)

Children with acute epiglottitis may develop acute respiratory obstruction and require intensive care management in form of:

- Endotracheal intubation or tracheostomy

- Oxygenation
- Humidification
- Oximetry

LARYNGITIS

Acute Laryngitis

Acute laryngitis is often associated with upper respiratory infection. It is usually viral in origin and presents with hoarseness of voice. Treatment is steam inhalation, analgesics and voice rest. It usually gets resolved in 2-3 weeks.

Chronic Laryngitis

If hoarseness of voice lasts for 3-4 weeks, patient should be referred to ENT surgeon particularly in smokers. Its cause can be:

Specific

Caused by

- Mycobacteria
- Fungal infection
- Syphilis

Treatment is specific for causative organism.

Non-specific

Caused by

- Smoking
- Sepsis of respiratory tract
- Voice abuse
- Gastro-esophageal reflux disease

Treatment is elimination of predisposing factors. In neglected cases, laryngeal mucosa may become dysplastic and premalignant.

Vocal Cord Polyp

It is a soft, grey, pedunculated mass on vocal cord, mostly unilateral. It is usually associated with smoking, voice abuse or acute infection. Treatment is removal by microdissection or laser surgery.

Laryngocele

See Chapter 12: Cysts and Neck Swellings.

VOCAL CORD PALSY

All the muscles of larynx are supplied by recurrent laryngeal nerve except cricothyroid muscle that is innervated by superior laryngeal nerve. Unilateral recurrent laryngeal nerve palsy leads to paramedian position of the affected vocal cord due to unopposed adducting action of cricothyroid muscle. Bilateral recurrent laryngeal nerve palsy leads to paramedian position of both vocal cords causing acute respiratory obstruction.

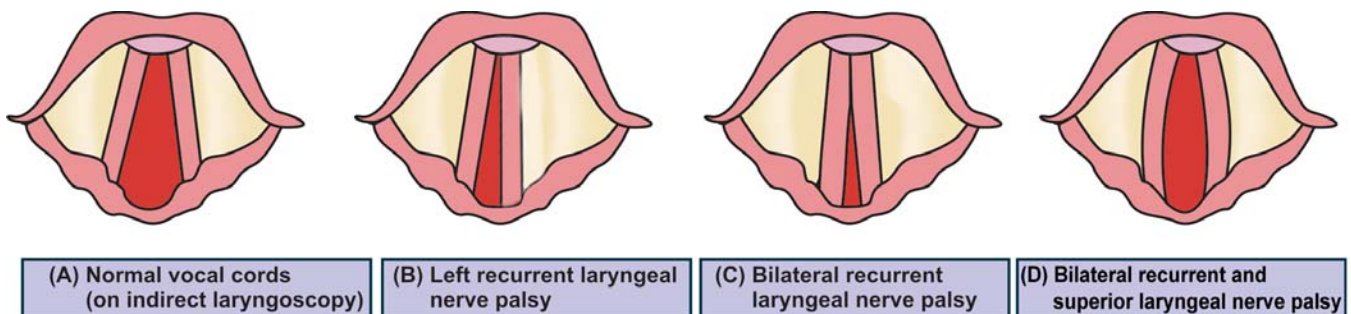
Palsy of both recurrent laryngeal nerve and superior laryngeal nerve (due to lesion of vagus nerve at high level) causes complete vocal cord paralysis that lies in "cadaveric position" (midway between abduction and adduction) (Figs 16.1A to D).

Etiology

It is given in Box 16.1.

Clinical Features

Unilateral recurrent laryngeal palsy is sudden in onset and presents with whispering voice. It may be associated with difficulty in swallowing liquids and weak cough reflex. Voice recovery may occur within a few weeks as



Figs 16.1A to D: Various vocal cord positions in recurrent laryngeal nerve palsy

Box 16.1: Etiology of vocal cord palsy

Traumatic	Thyroid surgery (commonest) Neck injury
Neoplastic	Carcinoma larynx Carcinoma thyroid Carcinoma esophagus Carcinoma lung involving left hilum
Infective	Viral infection
Vascular	Aortic aneurysm
Neurological	Lower motor neuron palsy

muscles of the opposite vocal cord move across the midline to meet the paralyzed vocal cord.

Bilateral recurrent laryngeal palsy is uncommon and seen as a serious complication of thyroid surgery on both lobes. On extubation following thyroidectomy, there is acute dyspnea and stridor. Patient requires immediate intubation or tracheostomy to prevent asphyxia.

Investigations

CT scan from skull base to diaphragm reveals most of the pathologies causing undiagnosed vocal cord palsy.

Treatment

In $\frac{1}{4}$ th cases, vocal cord palsy occurs without known pathology and spontaneous recovery occurs with conservative treatment.

In unilateral vocal cord palsy not recovering with conservative treatment, teflon paste is injected in the affected vocal cord so as to displace it medially. Alternatively, the vocal cord can be displaced medially by a surgical procedure (Thyroplasty).

In bilateral vocal cord palsy, tracheostomy is required immediately and that may need to be kept for six months to one year till recovery occurs. After that, surgery may be carried out to remove a portion of one arytenoid cartilage (Arytenoidectomy). It may be performed endoscopically using CO₂ laser.

TUMORS OF THE LARYNX**Benign Tumors**

These are extremely rare.

- a. *Papilloma*: It is the commonest benign tumor. It is probably caused by human papilloma virus. In adults,

it is usually single and presents as a pedunculated mass attached to vocal cords. The patient presents with hoarseness of voice. The diagnosis is made with laryngoscopic examination. The treatment is laser ablation or surgical excision since it may rarely become malignant. In children, papillomas are usually multiple with high tendency of recurrence. It is self-limiting condition and disappears spontaneously by adult life. Hence, it should not be subjected to radical excision for fear of damaging vocal cords.

- b. *Angiofibroma*: It is always single and presents as a small, smooth, red colored mass on the vocal cord. The patient presents with hoarseness of voice and hemoptysis. The diagnosis is made on laryngoscopic examination and the treatment is endoscopic removal or cryosurgery.

Malignant Tumors

Squamous cell carcinoma is the commonest tumor of larynx. It is the most common malignancy of the upper aerodigestive tract.

Incidence

It is most commonly seen in elderly male smokers. However, sex incidence is changing due to increased smoking habits among women. The male to female ratio has dramatically decreased from 10 : 1 to 5 : 1 in last two decades.

Etiology

- Exposure to tobacco (smoking) is most important etiological factor.
- Other likely cofactors are:
Metal dust (Nickel)
Wood dust
Asbestos
Hair dyes
- There is some unclear relation between adult onset papilloma and carcinoma larynx.

Classification

There are three varieties of laryngeal carcinoma based on its location:

- a. *Glottic*: It is the commonest variety. The tumor arises from true vocal cords involving anterior half. It is mostly papillary in appearance. Due to paucity of

lymphatic vessels in the vocal cords, it remains locally malignant for long time. The first symptom is hoarseness of voice that is progressive and may lead to stridor and aphonia. When tumor extends outside the glottis, it becomes aggressive and involves cervical lymph nodes. Due to slow growth, it has most favorable prognosis.

- b. *Subglottic*: It is rare variety. The tumor arises below vocal cords. The tumor grows steadily and silently till dyspnea develops. Hoarseness of voice indicates late disease. The growth may involve thyroid gland and deep cervical lymph nodes.
- c. *Supraglottic*: It is also called extrinsic laryngeal carcinoma and it involves false vocal cords, laryngeal ventricles and root of epiglottis. Due to abundant lymphatic supply, it presents with advanced stage disease and has worst prognosis. The patient presents with throat pain, hoarseness of voice and dysphagia. Neck nodes are involved in majority of the cases.

Staging

TNM staging of carcinoma larynx is given in Box 16.2.

Investigations

- Hopkins rod examination allows precise determination of extent of tumor.
- Direct laryngoscopy and biopsy confirms the diagnosis.
- CT and MRI are useful in determining the invasion of thyroid cartilage, suspicious nodal involvement in the neck and staging the disease.

Treatment

Early glottic and supraglottic tumors (stage I and II): are best treated with *mega voltage radiotherapy*. Dose is 60 Gy in 30 fractions over 6 weeks and cure rates are 90% and 70% in stage I and stage II respectively. Advantage is of voice preservation after the treatment. Alternative treatment for early tumors is excision by:

- *Endoscopic laser surgery*
- *Laryngofissure* in which thyroid cartilage is opened anteriorly in the midline and tumor removed under vision.
- *Partial laryngectomy*

However, voice result after surgery is not as satisfactory as that with radiotherapy.

Advanced Laryngeal Tumors

Treatment is *total laryngectomy*. It includes removal of entire larynx, hyoid bone, pre-epiglottic space, strap muscles and one or two tracheal rings with permanent tracheal stoma. When cervical lymph nodes are involved with secondary deposits, block dissection of lymph nodes is combined with laryngectomy.

Vocal Rehabilitation after Laryngectomy

For speech, vibrations are created in the pharynx by following ways:

- i. *Artificial larynx*: It is a battery powered device that is applied to the soft tissues of neck creating a primary sound while the patient articulates to produce words.
- ii. *Esophageal voice*: Patient swallows air into pharynx and upper esophagus. On regurgitation of air, pharyngo-esophageal mucosa vibrates to produce sound.
- iii. *Blom-Singer Valve*: A simple tracheo-esophageal puncture is maintained patent by a small tube containing a valve. This one way valve allows air to pass from trachea into the pharynx but prevents back flow of food particles into the airway. The air entering into pharynx and esophagus is modulated by tongue, lips and buccal mucosa to produce voice. 80% of the patients are able to develop fluent speech.
- iv. Larynx transplant has as yet been unsuccessful.

TRACHEOSTOMY

It is making an opening in anterior wall of the trachea and converting it into a stoma on skin surface.

Indications

1. *Upper airway obstruction*
 - Foreign body
 - Infection (diphtheria, Ludwig's angina)
 - Edema of glottis (head and neck burns)
 - Bilateral vocal cord palsy
 - Trauma (faciomaxillary, larynx, trachea)
 - Tumor (carcinoma larynx)
 - Congenital lesions (web, atresia)
 - Chronic stenosis (Tuberculosis, scalding)

Box 16.2: TNM staging of carcinoma larynx

Primary tumor (T)

T _x	Tumor cannot be assessed
T ₀	No evidence of primary tumor
T _{1s}	Carcinoma <i>in situ</i> .

Supraglottis

T ₁	Confined to site of origin with normal mobility.
T ₂	Tumor involves adjacent supraglottic site or glottis without fixation.
T ₃	Tumor limited to larynx with extension to post-cricoid area, medial wall of pyriform sinus or pre-epiglottic space.
T ₄	Tumor extends beyond larynx to involve oropharynx, soft tissues of neck.

Glottis

Tumor confined to vocal cords with normal mobility.
Supraglottic or subglottic extension with normal or impaired cord mobility.
Tumor confined to larynx with cord fixation.
Tumor extends beyond larynx to involve oropharynx, soft tissues of neck.

Subglottis

Tumor confined to subglottic region.
Tumor extension to vocal cords with normal or impaired cord mobility.
Tumor confined to larynx with cord fixation.
Tumor extends beyond larynx to involve oropharynx, soft tissues of neck.

Regional lymph nodes (N)

N _x	Lymph nodes cannot be assessed.
N ₀	No clinically positive nodes.
N ₁	Single homolateral node 3 cm or less in diameter.
N _{2a}	Single homolateral node 3-6 cm in diameter.
N _{2b}	Multiple homolateral nodes 3-6 cm in diameter.
N ₃	Massive nodes (>6 cm)

Distant metastasis (M)

M _x	Metastasis cannot be assessed.
M ₀	No distant metastasis.
M ₁	Distant metastasis present.

Stage grouping

Stage I	T ₁ N ₀ M ₀
Stage-II	T ₂ N ₀ M ₀
Stage III	T ₁ N ₁ M ₀ , T ₂ N ₁ M ₀ , T ₃ N ₁ M ₀
Stage IV	T ₄ N ₀ M ₀ , Tany N ₂ M ₀ , Tany Nany M ₁

2. *Retained secretions*

- Severe bronchopneumonia
- Chronic bronchitis
- Chest injury (Flail chest)

3. *Respiratory insufficiency*

- Head injury
- Bulbar poliomyelitis

- Barbiturate poisoning
- Tetanus

Aims of Tracheostomy

Aim is to assist respiration which it does in the following ways:

- i. It relieves upper airway obstruction.

- ii. It reduces the anatomical dead space (150 ml).
- iii. Toilet of tracheobronchial tree by giving direct access.
- iv. Cuffed endotracheal tube protects the airways from aspiration and allows positive pressure ventilation to be maintained for a prolonged period.

However, all these objectives can be achieved, to some extent, by the use of endotracheal tube. But prolonged endotracheal intubation risks laryngeal damage and subglottic stenosis. Hence, tracheostomy is indicated when endotracheal intubation fails in emergency situations or prolonged ventilation is required (more than a week) in elective situations.

Advantages of tracheostomy over endotracheal intubation are:

- i. Patients are more comfortable and require no sedation.
- ii. It can be continued indefinitely.
- iii. Suction and clearing of secretions is easier.
- iv. Work of breathing is reduced.
- v. Alveolar ventilation is increased.
- vi. Weaning is easier with tracheostomy.

Disadvantages of tracheostomy are:

- i. It is an open wound liable to infection.
- ii. Loss of heat and moisture leading to desiccation and metaplasia of tracheal epithelium.
- iii. Tracheostomy tube acts as a foreign body that stimulates mucus production in the trachea. The mucus gets encrusted and blocks the tube.

Types of Tracheostomy

- i. *Emergency*: It is done for acute airway obstruction. If facilities don't exist and experienced doctor is not available, a large intravenous cannula may be inserted into cricothyroid membrane to relieve acute upper airway obstruction.
- ii. *Elective*: During certain operations on upper airway.
- iii. *Permanent*: Following laryngectomy.

Surgical Anatomy

The trachea begins as a continuation of the larynx at lower border of cricoid cartilage. It is superficial in the upper part and it becomes more deeply placed as it

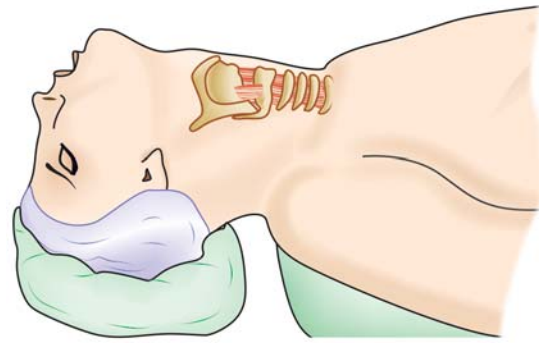


Fig. 16.2: Position of the patient for tracheostomy

descends. Its superficial relations include skin, platysma, investing layer of deep fascia, strap muscles (sternohyoid and sternothyroid), pretracheal fascia and isthmus of thyroid gland (overlies 2nd, 3rd and 4th tracheal rings). The tissue planes in the midline are devoid of major veins.

Operation

a. *Emergency Tracheostomy*

Patient is placed in supine position. Neck is extended by placing pillow between shoulders (Fig. 16.2). However, in a case of severe head and neck trauma with suspected cervical spine injury, it is safer to do cricothyroidotomy rather than tracheostomy. Local anesthesia is infiltrated (not required in deeply unconscious patient). 1-1½" vertical skin incision is given below cricoid cartilage in the midline (Fig. 16.3). Skin, platysma, deep fascia and pretracheal fascia are divided passing between infrahyoid muscles. If isthmus comes in the way, it is ligated and divided. A cricoid hook is then inserted under the cricoid cartilage and pulled up to stabilize the trachea and to bring it to the surface. The second, third and fourth tracheal rings are divided with a knife (Fig. 16.4). A trachea wound dilator is inserted to dilate the tracheal wound. A tracheostomy tube is then inserted into the trachea and dilator is removed (Fig. 16.5). Air movements through tracheostomy tube opening are felt with fingers to check its correct position. If tube is not placed correctly, it will lead to surgical emphysema and respiratory obstruction (Figs 16.6A and B). The cuff of tracheostomy tube is inflated to make it self-retaining. The tube

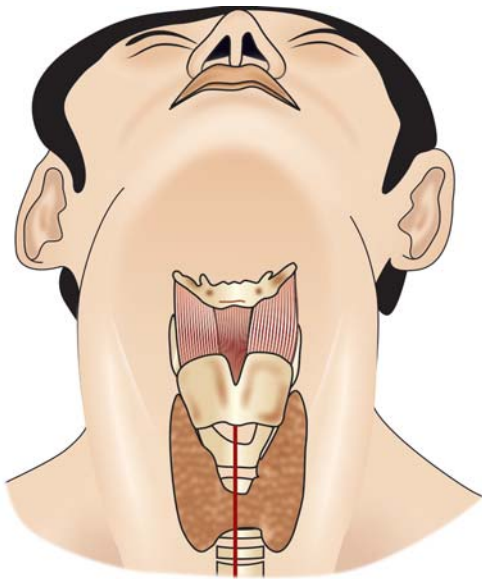


Fig. 16.3: Emergency tracheostomy—vertical skin incision

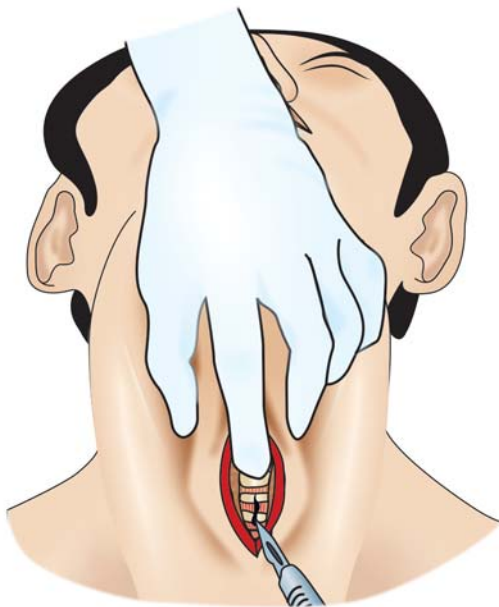


Fig. 16.4: Emergency tracheostomy—incising the trachea

is attached with tapes that are tied around patient's neck (Figs 16.7 and 16.10). Alternatively, the flanges of the plastic tube may be stitched directly to the underlying skin. The wound should be sutured lightly to prevent surgical emphysema.

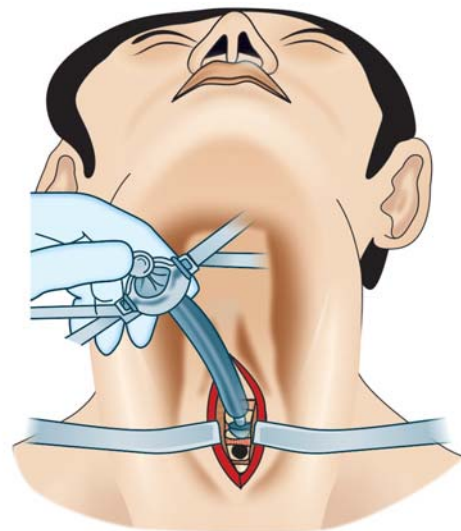
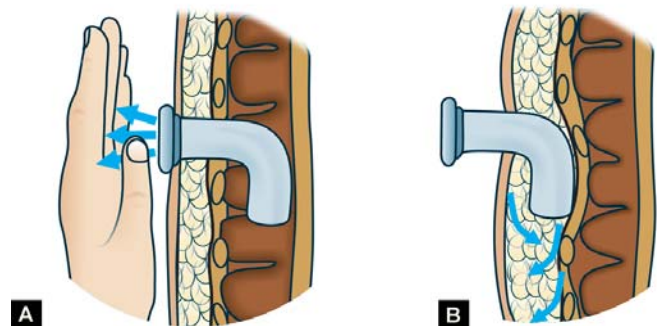


Fig. 16.5: Emergency tracheostomy—inserting the tracheostomy tube



Figs 16.6A and B: Checking position of tracheostomy tube—(A) Tracheostomy tube in correct position, (B) Misplaced tracheostomy tube causing surgical emphysema



Fig. 16.7: Tracheostomy tube secured in a patient of head injury with fracture mandible

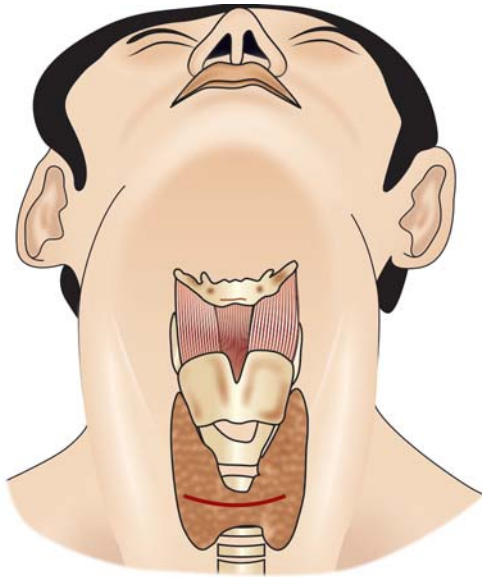


Fig. 16.8: Elective tracheostomy—transverse skin incision

b. Elective Tracheostomy

The advantage of elective tracheostomy is that there is complete airway control, precise dissection and careful placement of appropriate tube.

After positioning, local or general anesthesia is given. A transverse skin incision is given over third tracheal ring (it heals with less scarring) (Fig. 16.8). If performed under local anesthesia, injection of 2% xylocaine into trachea before incising prevents violent cough following insertion of the tube. An inverted U-shaped incision is made on second and third tracheal rings to raise a flap (Bjork flap). The tip of the flap is stitched to the inferior edge of the transverse skin incision (Fig. 16.9). Its advantage is that it prevents tube displacement and allows reintroduction of displaced tube with minimum difficulty.

Aftercare of Tracheostomy

- i. *Humidification:* Warm, wet, oxygenated air is flown over the stoma to make tracheal secretions less viscid.
- ii. *Clearance of secretions:* Intermittent suction is done at regular intervals to keep the tracheo-bronchial tree free from secretions. Strict asepsis should be maintained by keeping suction

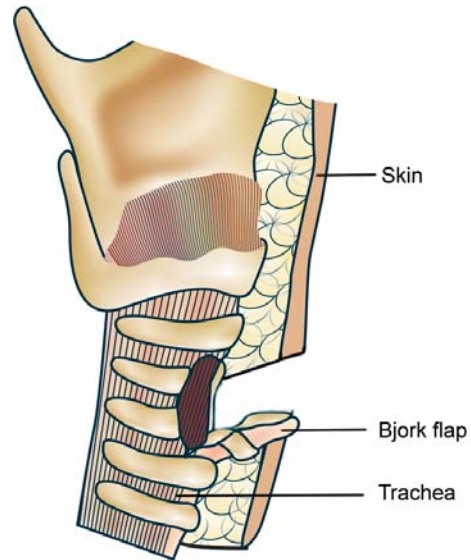


Fig. 16.9: Bjork flap in elective tracheostomy

catheter in a sterile holder. The catheter is introduced with aseptic conditions after wearing mask and gloves.

- iii. *Clearance of thick mucus:* When mucus is very thick and difficult to aspirate, isotonic saline, sodabcarb or mucolytic agent should be administered through the tracheostomy tube by a fine nebulizer. If there is inner tube, it should be removed and washed in sodabcarb solution.
- iv. *Care of cuff:* Low pressure cuff should be used so that it does not occlude the mucosal circulation. High pressure cuff can necrose the cartilage and can cause tracheal collapse.
- v. *Replacement of tube:* Tube should be replaced every 3-4 days until a tract is established. During replacement one should be careful to place the tube correctly in tracheal lumen. A good airflow is apparent if the tube is in correct place.

Complications of Tracheostomy

Intraoperative Complications

- Hemorrhage
- Recurrent laryngeal nerve injury
- Tracheal injury
- Esophageal injury

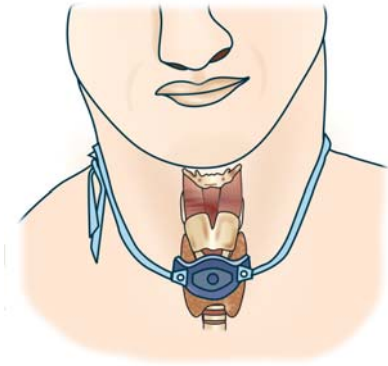


Fig. 16.10: Tracheostomy tube in position with tapes around patient's neck

Postoperative complications

- Surgical emphysema
- Pneumothorax
- Pneumomediastinum
- Aspiration pneumonia
- Accidental dislodgement of the tube
- Wound infection
- Tracheal stenosis
- Tracheo-esophageal fistula
- Tracheo-cutaneous fistula
- Tracheo-innominate artery fistula (severe hemorrhage).

17

Chapter

Head Injury and Cranial Nerves Injury

Sanjay Marwah

- Head injury accounts for one-third of all trauma deaths.
- It is the leading cause of death and disability in young adults.
- The principal causes of head injury are road traffic accidents, assaults, falls, sport injuries and industrial accidents.
- *Acceleration/Deceleration injuries*: Due to mass movement of brain within closed cranial cavity.
- *'Coup' injury*: Brain injury occurring at the site of blow.
- *'Contre-coup' injury*: Injury to the brain surface opposite to the site of blow.

PATHOPHYSIOLOGY OF HEAD INJURY

Primary Injury

It is the damage occurring at the time of initial impact. It consists of diffuse axonal injury and focal contusions. It is not treatable and can only be prevented, e.g. by wearing seat belts and crash helmets.

Secondary Injury

It is the additional insult imposed on normal tissue following primary injury (Box 17.1). The focus of medical management is to prevent the secondary damage.

Box 17.1: Causes of secondary brain damage

- Hypotension
- Hypoxia
- Hyperthermia
- Convulsions
- Raised intracranial pressure
- Hyperglycemia

MECHANISMS OF HEAD INJURY

- *Closed injury*: Due to blunt trauma.
- *Open injury*: Due to penetrating trauma, there is communication between intradural contents and atmosphere.

CLASSIFICATION OF HEAD INJURY

Anatomically, head injury can be classified starting from skin and going to the brain (Box 17.2).

Box 17.2: Classification of head injury

Scalp	Laceration, contusion
Skull	Fracture (simple, comminuted, depressed, compound)
Dura	Laceration
Brain	
<i>Primary injury:</i>	Diffuse axonal injury, concussion, contusion, laceration
<i>Secondary injury:</i>	Edema, ischemia, hematoma, coning, infection, epilepsy
Blood vessels	Extradural, subdural, intracerebral or intraventricular bleed Supra/Infra-tentorial bleed
CSF	CSF rhinorrhea/otorrhea Infection (meningitis) Obstruction (hydrocephalus)
Cranial nerves	Avulsion, compression
Associated injuries	To eye, ear, paranasal sinuses, cervical spine

SCALP LACERATION

- It causes profuse bleeding because of:
 - Rich vascularity

- Blood vessels lie in dense fibrous layer superficial to epicranial aponeurosis and remain open once transected.
- In infants, scalp bleeding may cause hypovolemic shock.
- The deep lacerations in the scalp should always be palpated with gloved finger for any evidence of depressed fracture.
- The scalp laceration should be repaired as follows:-
 - Shaving of hair adjacent to wound.
 - Apply soap on adjoining hair so that they get matted and do not fall in clean area.
 - Clean the wound.
 - Intradermal injection of 1% lignocaine for local anesthesia.
 - Trimming of devitalized skin tags.
 - In case of fresh bleeding, apply artery forceps on galea deep to artery and evert skin edges.
 - Apply interrupted skin stitches including a bite of galea so as to control bleeding.
- Although rare, infection can occur deep to galea and it spreads rapidly due to presence of loose areolar tissue. Infection can reach intracranial sinuses through emissary veins. Osteomyelitis of skull is associated with subperiosteal swelling and edema of scalp called as '**Potts' puffy tumor**' (Box 17.3A).

SKULL FRACTURES

Head injury can cause following types of skull fractures:-

- i. *Simple linear fracture*: It is the most common type of fracture and indicates severe head injury. A linear fracture of squamous temporal bone may lacerate middle meningeal artery and can cause extradural hematoma. Such patient should be hospitalized and closely observed for 48 hours. A

Box 17.3A: Pott's puffy tumor

- Subperiosteal infection of vault.
- Cause—osteomyelitis of skull, infected subperiosteal hematoma.
- Dumb bell abscess—pus in subperiosteal space and extradural space communicating with each other.
- Pitting edema of scalp.
- Severe headache, vomiting, blurred vision.
- CT scan is diagnostic.
- Treatment:
Burr hole and pus drainage.
Antibiotics.

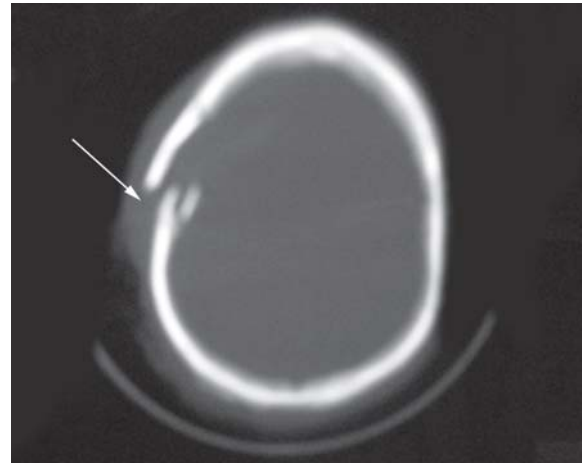


Fig. 17.1: CT scan of the head showing depressed fracture of skull

Box 17.3B: Complications of depressed fracture

- Dural tear
- Pneumocranium
- Intracranial hematoma
- Infection (meningitis) in compound fracture
- Epilepsy
- Cosmetic deformity
- Raised intracranial pressure (due to pressure on venous sinuses)

linear fracture on skull X-ray can be confused with vascular markings and suture lines.

- ii. *Depressed fracture*: It is considered significant if on skull X-ray/CT scan, degree of depression is greater than depth of inner table of skull (Fig. 17.1). The complications likely to be seen in depressed fracture are shown in Box 17.3B.

In infants and children, depressed fracture is seen as concave depression of the skull and is called as **Pond fracture** (Figs 17.2A and B).

In compound depressed fracture causing dural tear, there is risk of CSF leak and air entering into the cranial cavity (pneumocranium) (Fig. 17.3). Here, the scalp wound should be debrided, bone fragments elevated and dural tear repaired.

- iii. *Base of skull fracture*: It is usually not evident on routine skull X-ray and is diagnosed on clinical grounds.

Anterior fossa fractures present with:

- CSF rhinorrhea—if nasal discharge contains glucose, then the fluid is CSF and not the mucin.



Fig. 17.2A: Pond fracture of skull in an infant

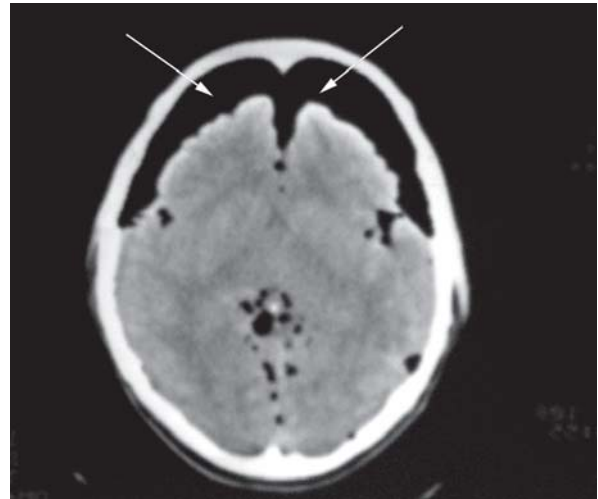


Fig. 17.3: CT scan head showing massive pneumocranium of anterior cranial fossa



Fig. 17.2B: X-ray skull showing Pond depressed fracture



Fig. 17.4: 'Raccoon' eye

- 'Raccoon' or 'panda' eyes—there is periorbital hematoma limited to orbital margin. It indicates subgaleal hemorrhage that tracks down in eyelids (Fig. 17.4). Also there is subconjunctival hemorrhage extending beyond posterior limit of sclera indicating bleed tracking down from orbital cavity.
- I, III, IV and V cranial nerves may be involved.

Middle fossa fractures present with:

- CSF otorrhea.
- VII and VIII cranial nerve palsy.

Posterior fossa fractures present with:

- Battle's sign—bruising over the mastoid (behind the ear) that develops 24-48 hours after injury.

- IX, X and XI cranial nerves may be involved.

In base of skull fractures, there is potential risk of meningitis due to CSF leak, so prophylactic antibiotics should be given.

BRAIN INJURY

It can be primary or secondary brain injury.

Primary Brain Injury

It is the injury occurring at time of impact. The various types are:

- Diffuse axonal injury:* It is due to shearing at junction of grey matter with white matter. Its severity may

range from mild damage causing confusion to severe damage causing coma and even death.

- b. *Cerebral concussion*: It literally means “to shake”. There is brief temporary paralysis of function without organic structural damage. The patient has transient loss of consciousness that recovers completely after a variable period of time. In most cases, there is amnesia for the event of injury.
- c. *Cerebral contusion*: It indicates more severe injury. There is bruising and edema of the brain. The patient has focal neurological deficit that may resolve or persist.
- d. *Cerebral laceration*: The brain surface is torn and there is intracerebral hemorrhage. The patient presents with focal neurological deficit.

Cerebral contusion and laceration are seen as areas of bleeding on CT scan.

Secondary Brain Injury

It is a consequence of primary brain injury and consists of:

- a. *Cerebral edema*: It can be localized or diffuse edema. It results in raised intracranial pressure leading to deterioration in level of consciousness. It is more common in children and causes severe damage.
- b. *Cerebral ischemia*: It is due to rise in intracranial pressure leading to impaired cerebral perfusion and cerebral hypoxia.
- c. *Intracranial hematoma*: It is due to arterial or venous bleeding.
- d. *Coning or cerebral herniation*: Due to rise in intracranial pressure, there is herniation of brain through tentorial hiatus or foramen magnum leading to rapid deterioration and irreversible brain damage (see below).
- e. *Infection*: It is seen in compound fractures where infection enters the central nervous system leading to meningitis and brain abscess (Box 17.4). The patient presents with fever and neck stiffness. If foreign bodies are retained following penetrating trauma, chances of infection become very high (Fig. 17.5).
- f. *Epilepsy*: It is due to brain injury and edema. It is more common in children. It may cause rapid deterioration in level of consciousness.

INJURY TO BLOOD VESSELS

It leads to intracranial hemorrhage causing brain compression. The severity of brain compression depends upon:

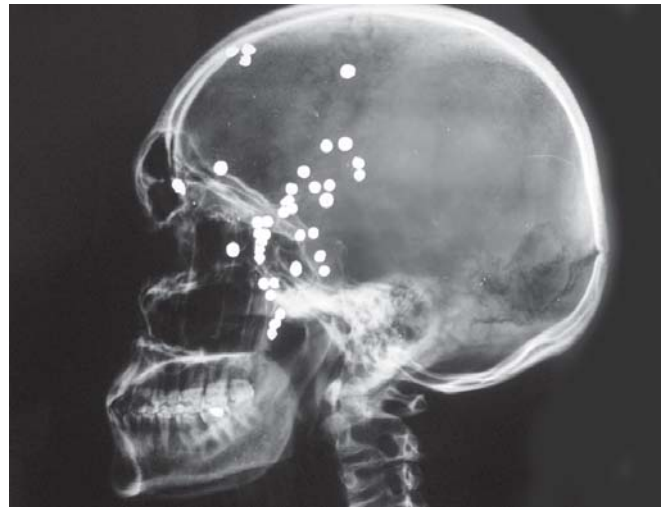
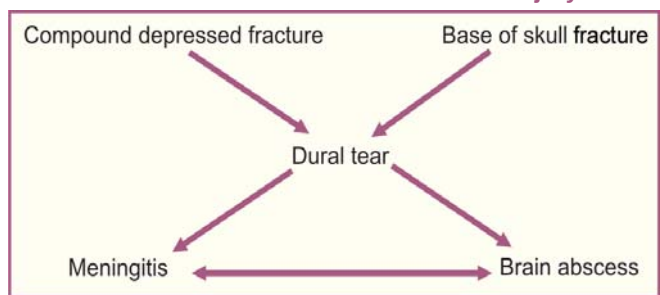


Fig. 17.5: X-ray skull showing multiple pellets following fire-arm injury. The patient developed brain abscess

Box 17.4: Cerebral infection in head injury



- Size and number of bleeding vessels
- Compartment of bleeding
- Plane of bleeding.

A. Compartment of Bleeding

Surgical Anatomy of Intracranial Compartments

Tentorium cerebelli divides cranial cavity into supratentorial and infratentorial compartments. The former contains cerebral hemispheres and latter contains cerebellum, pons and medulla. Two compartments are continuous with each other through tentorial hiatus. Midbrain passes through this hiatus. Important structures in midbrain are cerebral peduncles, oculomotor nerves and reticular formation. The reticular formation controls consciousness by its interaction with cerebral cortex. Uncus of temporal lobe lies immediately above and lateral to tentorial hiatus. The motor fibers cross in the brainstem and go to opposite side of spinal cord (Fig. 17.6).

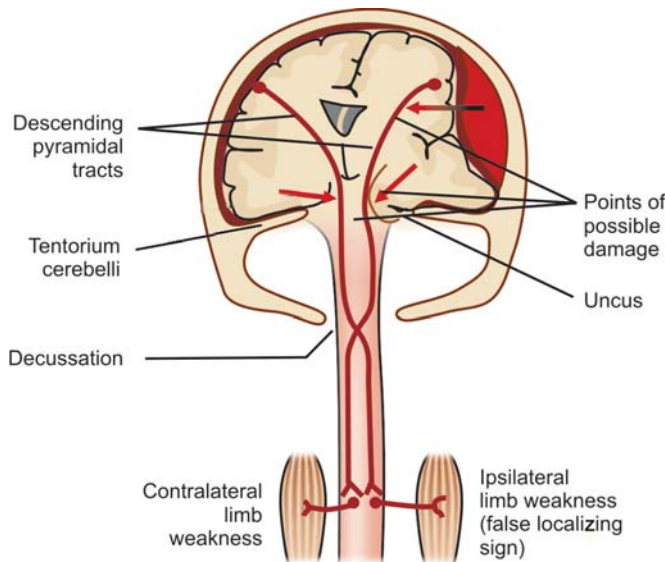


Fig. 17.6: Effects of extradural hematoma

Supratentorial Hemorrhage

Its effects are partly by local pressure on brain underlying the hematoma, but more important by herniation of uncus of temporal lobe through tentorial hiatus causing midbrain compression (Fig. 17.6). The effects of midbrain compression are:

- Deterioration in conscious state due to compression of reticular formation.
- Pupillary changes due to stretching of oculomotor nerves (**Hutchinson's pupils**). Initially, there is transient constriction of pupil on affected side due to irritation of oculomotor nerve followed by dilatation due to paralysis of the nerve. As compression becomes more severe, similar pupillary changes are seen in the opposite eye due to stretching of opposite side nerve.
- Hemiparesis due to compression of cerebral peduncle by the edge of tentorium cerebelli. In most cases, hemiparesis occurs in the limbs contralateral to the side of lesion due to crossing of the fibers. Sometimes, hemiparesis occurs on side of hematoma due to indentation of contralateral cerebral peduncle by the edge of tentorium cerebelli (**Kernohan's notch**).

With continuing compression and damage to pons, following signs appear:

- Rise in blood pressure

- Bradycardia
- Irregular respiration

Finally, impaction of midbrain cone (herniation) produces irreversible brain damage in form of fixed dilated pupils and decerebrate rigidity.

Infratentorial Hemorrhage

It causes compression of cerebellar hemisphere, pons, medulla and lower cranial nerves. Midbrain is not initially compressed, so consciousness is not impaired. The effects of infratentorial hemorrhage are:

- Irregular respiration
- Rise in blood pressure
- Bradycardia
- Ataxia
- Lower cranial nerves palsy.

Infratentorial hemorrhage is far less common than supratentorial hemorrhage.

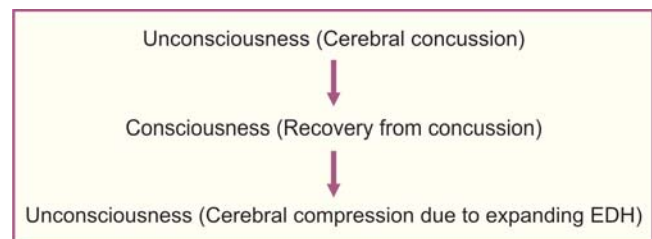
B. Plane of Bleeding

i. Extradural Hematoma (EDH)

It is the commonest cause of intracerebral hematoma in young adults (Box 17.6). It is commonly due to laceration of middle meningeal artery following temporal bone fracture as a result of blow on side of the head. It can also occur due to laceration of dural venous sinuses.

Lucid interval is a characteristic finding in extradural hematoma. After initial period of unconsciousness due to cerebral concussion, there is a period of consciousness (Lucid interval) and again patient becomes unconscious in a variable period of time due to cerebral compression caused by EDH (Box 17.5). In middle meningeal hemorrhage, as clot enlarges in size and exerts pressure on underlying cerebral cortex, the paralysis starts in face and then affects the arm and proceeds towards leg

Box 17.5: Lucid interval



(*March of paralysis*). If urgent decompression of the clot is not done, irreversible brain damage occurs due to midbrain coning.

ii. Subdural Hematoma (SDH)

There is bleeding in subdural space (Box 17.6).

a. Acute Subdural Hematoma

It is seen less commonly. There is cortical laceration or bleeding from dural venous sinuses. It progresses very rapidly and may lead to death within hours if early evacuation is not done.

b. Chronic Subdural Hematoma

It is more common. It is due to venous bleeding caused by rupture of bridging veins from surface of cerebral cortex to the dura. It is commonly seen in elderly people with cerebral atrophy. It results from mild or even unnoticed head injury that results in slow collection of blood in subdural space. The clinical features are progressive neurological deficit and fluctuating level of consciousness starting in 2-3 weeks time. If not suspected, the pathology may be mistaken as senile dementia or brain tumor. It is diagnosed on CT scan and treatment is evacuation of hematoma.

Box 17.6: Comparison of EDH and SDH

	EDH	SDH
Age	Young	Elderly
Bleeding vessels	Arterial (middle meningeal artery)	Venous (Subdural veins)
Onset	2-12 hours	2 weeks
Lucid interval	Present	Absent
Hutchinson's pupil	Present	Absent
March of paralysis	Present	Absent
X-ray skull	Fracture seen crossing groove of middle meningeal artery	Fracture crossing middle meningeal artery not seen
CT head	Biconvex density (Fig. 17.7)	Concavo-convex density (Fig. 17.8)

iii. Intracerebral and Intraventricular Hemorrhage

It is seen in severe head injury due to cerebral contusion and laceration (Fig. 17.9).

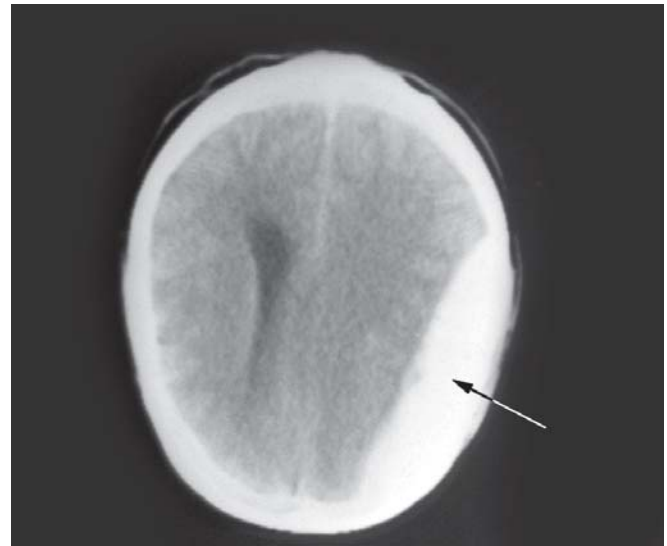


Fig. 17.7: CT head showing large extradural hematoma (biconvex) with ventricular effacement and midline shift

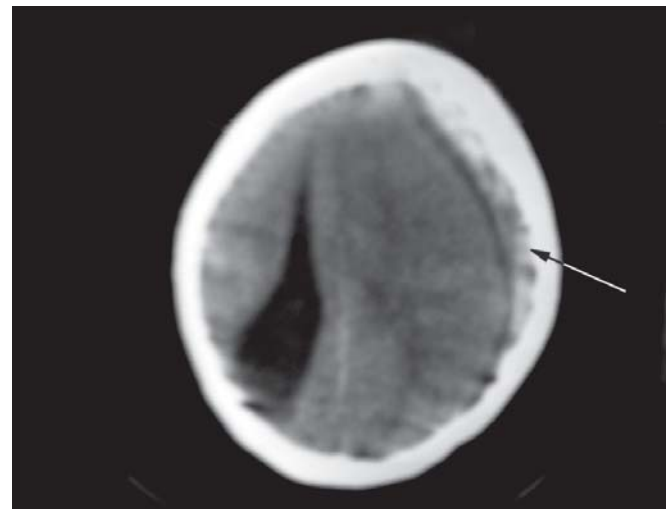


Fig. 17.8: CT head showing subdural hematoma (concavo-convex) with midline shift

MANAGEMENT OF HEAD INJURY PATIENT

- Initial management should follow the Advanced Trauma Life Support (ATLS) guidelines (see chapter 10: Care of the Acutely Injured).
- Establishment of airway, breathing and circulation are the first priorities.
- All patients of head injury should be assumed to have simultaneous cervical spine injury until proven otherwise. So cervical immobilization should be done with a cervical collar.

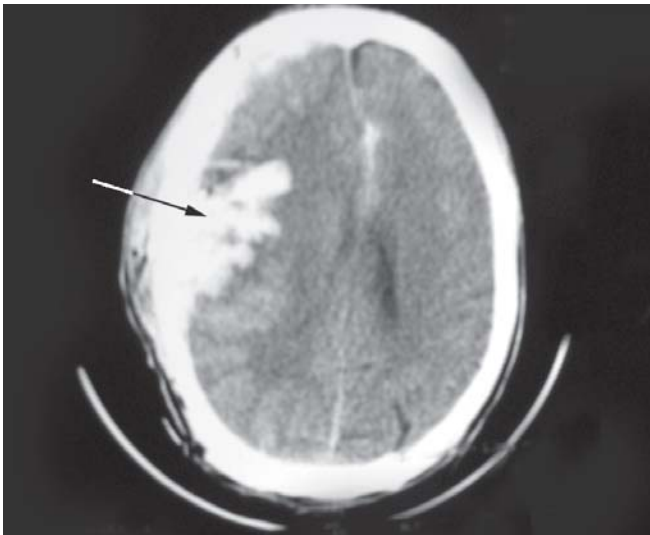


Fig. 17.9: CT head showing intracerebral hemorrhage

- Life-threatening extracranial injuries (e.g. chest and abdominal injuries) take priority over intracranial injuries and should be managed first.
- *Neurological assessment:*
 - a. *Level of consciousness* is best assessed by Glasgow Coma Scale (Box 17.7).

Box 17.7: Glasgow Coma Scale (GCS)

Motor function	Score
Obedient commands	6
Localizing pain	5
Flexion	4
Abnormal flexion	3
Extension	2
None	1
Verbal response	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Eye opening	
Spontaneous	4
To speech	3
To pain	2
None	1

The parameters seen are eye opening, verbal and motor response. It gives maximum score of 15 (fully conscious) and a minimum score of 3 (coma). After initial recording, patient should be frequently reassessed (every 15-30 minutes) to look for deterioration or improvement in level of consciousness. GCS score helps in deciding management guidelines (Box 17.8).

Box 17.8: Management on basis of GCS

GCS	Severity of head injury	Management
3-8	Severe	Admit in ICU, Ventilatory support, CT scan
9-13	Moderate	Manage in A and E deptt, urgent CT scan
14-15	Mild	Evaluate, observe and discharge if no abnormality

- a. *Pupillary response:* Function of oculomotor nerve is the most useful indicator of an expanding intracranial lesion (see Hutchinson's pupil).
- b. *Limb weakness:* It is seen by comparing the response in each limb to painful stimuli.
- c. *Examination of cranium:*
 - Any scalp and facial wounds.
 - CSF rhinorrhea or otorrhea.
 - 'Raccoon' eyes.
 - Mandibular or facial fracture.
- d. *Vital signs:* Pulse, BP, respiration and temperature.
- e. *Examination of cranial nerves* (see below).
- f. *History of injury:*
 - History of loss of consciousness.
 - Period of loss of consciousness.
 - Period of post-traumatic amnesia.
 - Cause and circumstances of injury.
 - Presence of headache, vomiting and convulsions.
- g. *Indications of hospitalization:* See Box 17.9.
- h. *Indications for skull X-rays:* With easy availability of CT scan, indications of skull X-rays have diminished. However, X-rays still acts as useful guide in mild head injury cases and in remote areas where CT scan facilities are not available (Box 17.10).
- i. *Indications for CT scan:* CT scan is the gold standard investigation for head injury (Box 17.11). If CT scan

Box 17.9: Indications for hospitalization

On history

- Transient loss of consciousness
- Post-traumatic amnesia

On examination

- Altered level of consciousness
- Focal neurological signs
- CSF leak
- Coma

On X-ray

- Skull fracture

Box 17.10: Indications for skull X-rays

- History of loss of consciousness or amnesia.
- GCS of 14 or less.
- Persistent headache and vomiting.
- High energy mechanism of injury.
- Scalp swelling/laceration.
- Significant maxillofacial injury.

shows no abnormality, the patient should be observed for at least one night (Fig. 17.10). When an intracranial abnormality is revealed on CT scan, patient should be transferred to a neurosurgical unit for further management.

Box 17.11: Indications for CT scan

- Unconscious patient
- History of convulsions
- Difficulty in assessment (very young/very old/intoxicated)
- Focal neurological deficit
- Battle sign
- 'Raccoon' eyes
- CSF leak
- Confusion persisting after resuscitation (GCS <14)
- X-ray skull showing fracture
- Deterioration in level of consciousness

• *Medical management:*

- Correct hypovolemia by I/V fluids (dextrose saline). However, circulatory overload should be avoided as it can aggravate cerebral edema.

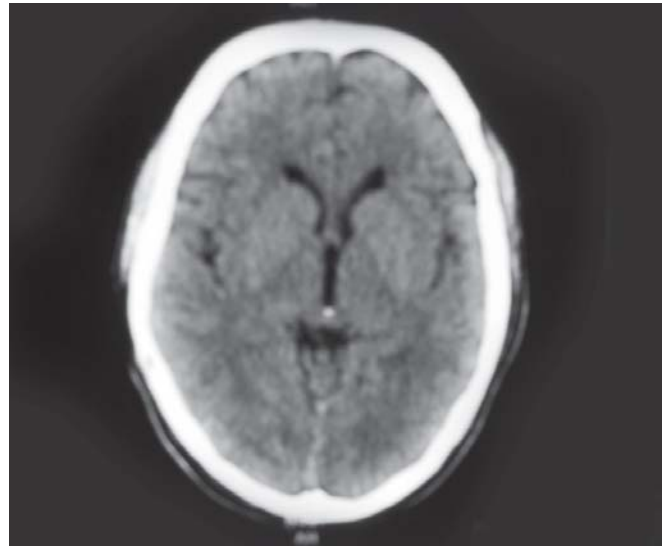
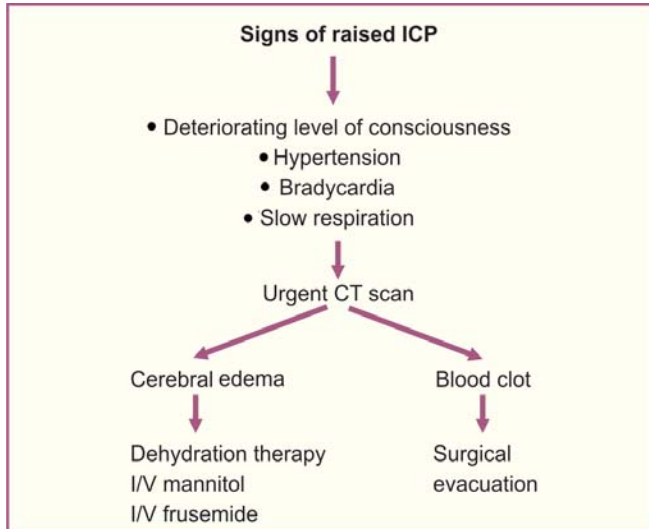


Fig. 17.10: Normal CT scan head

- Epilepsy causes rapid deterioration by raising the intracranial pressure. Bolus of I/V diazepam (0.1 mg/kg) controls epilepsy. Then phenytoin is given in dose of 100 mg 8 hrly.
- Electrolyte disturbances are common in severe head injury and must be corrected.
- Fever is another important cause of deterioration since it increases cerebral metabolism. It is controlled by hydrotherapy and antipyretics (paracetamol).
- Infection and meningitis are more likely in compound skull fractures with CSF leak. Broad spectrum antibiotics should be given prophylactically in such cases.
- Pain is a problem in conscious patient having other injuries (e.g. chest and limb injuries). Non-narcotic analgesics like diclofenac sodium should be given round the clock.
- Gastric ulceration (Cushing's ulcers) is known to cause upper GI bleeding in cases of head injury. Prophylactic proton pump inhibitors (omeprazole) should be given for its prevention.
- Steroids—it is now well-established that steroids have no benefit in management of head injury.
- If rise in intracranial pressure (ICP) occurs due to cerebral edema, it is managed by I/V mannitol or I/V frusemide (Box 17.12).
- In unconscious patient, management includes:

- Care of bladder - by catheterization.
 Care of bowel - by laxatives, enema.
 Care of back - by regular change of posture,
 using air or water mattress.
 Feeding by nasogastric tube/feeding jejunostomy.

Box 17.12: Management of raised ICP

- **Surgical management:** Emergency burr holes are required if patient suddenly deteriorates due to intracranial clot. In such situation, there may be no time to obtain neurosurgical help and patient's life can be saved by prompt evacuation of clot. Surgical steps are:
 - 3 cm vertical incision above mid point of zygoma down to bone.
 - Temporal bone is perforated with a burr.
 - Bone pieces removed with a bone nibbler and dura exposed
 - 'Black current jelly' clot over dura is removed and spurting middle meningeal artery secured with cautery or clip.
 - If there is no EDH and dura appears tense and bluish, it is due to SDH.
 - Incise the dura and drain the clot.
- **Delayed effects of head injury:** are given in Box 17.13.

CRANIAL NERVES**Classification of Nerve Injuries***Seddon Classification*

Nerve injuries are classified according to severity of injury (Box 17.14).

Box 17.13: Delayed effects of head injury

- Post-traumatic epilepsy
- Post-traumatic headache
- Post-traumatic hydrocephalus
- CSF fistula
- Neuro-psychiatric problems (Schizophrenia)
- Post-concussion symptoms (Insomnia, irritability, defective memory, lack of concentration)

Box 17.14: Classification of nerve injuries

<i>Neuropraxia</i>	<i>Axonotmesis</i>	<i>Neurotmesis</i>
Nerve fibers intact	Nerve fibers divided	Nerve fibers divided
Nerve sheath intact	Nerve sheath intact	Nerve sheath divided
Recovery complete	Near complete recovery that takes very long	Partial recovery if cut ends are approximated

Neuropraxia

It is equivalent to concussion and is a mild type of nerve injury. Nerve fibers as well as nerve sheath remain intact. There is local block to conduction of nerve impulse. There is temporary loss of sensations and muscle weakness. It is caused by nerve stretching (e.g. tourniquet, postoperative) and complete recovery occurs once cause is removed.

Axonotmesis

There is anatomical disruption of nerve fibers within intact nerve sheath. It results from more severe injury to the nerve, e.g. facial nerve palsy in fracture middle cranial fossa. Clinically, there is widespread loss of sensations, power and reflexes. Incomplete recovery takes place by downgrowth of axons within intact sheath. The rate of axonal growth is very slow (1 mm/day). If course of nerve is lightly percussed from below upwards, a tingling sensation is felt by the patient at site of regeneration (**Tinel's sign**).

Neurotmesis

Nerve is completely severed and spontaneous recovery is not possible. It is usually caused by penetrating wounds (e.g. stab wound, gunshot wound). If the nerve is left divided, there is formation of stump neuroma and

recovery does not occur. If surgical repair is carried out, partial recovery occurs by axonal regeneration. However, quality of recovery is not as good as in axonotmesis because cross union between sensory and motor fibers may occur.

Examination of Cranial Nerves

Olfactory Nerve (I)

Test perception of smell.

Optic Nerve (II)

Test for the vision by asking the patient to read, to count fingers or to differentiate light from darkness by throwing light on covered and uncovered eyes.

Oculomotor (III), Trochlear (IV) and Abducent (VI) Nerves

- Look at the pupils and note their size and shape.
- Look reaction of pupils to the light.
- Test for the ocular movements by asking patient to look to the left, right, upwards and downwards. Functions of various extraocular muscles are shown in Figure 17.11.
- In *oculomotor nerve paralysis*, there is:
 - ☐ Dilated pupil that does not constrict with light.
 - ☐ Drooping of upper eyelid (ptosis).
 - ☐ Impaired eye movements (inability to move the eyeball inwards or upwards—medial rectus and Superior rectus paralyzed).
- In *trochlear nerve paralysis*:
 - ☐ Downward and outward movement of eyeball is impaired (Superior oblique paralyzed).

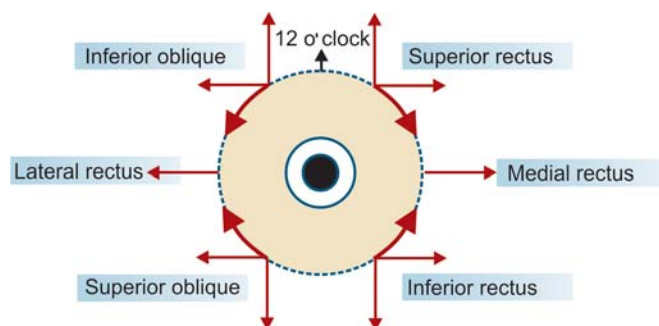


Fig. 17.11: Action of extraocular muscles indicated by arrows

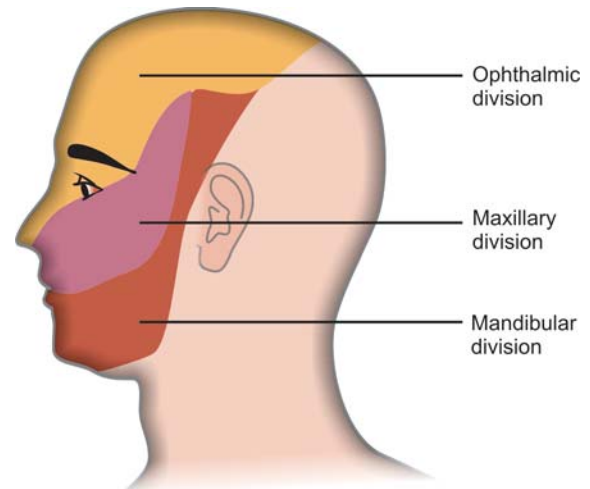


Fig. 17.12: Areas of sensory distribution of divisions of trigeminal nerve

- In *abducent nerve paralysis*:
 - ☐ There is internal squint and outward movement of eyeball is impaired (Lateral rectus paralyzed).

Trigeminal Nerve (V)

Motor function: Ask the patient to clench his teeth and feel masseter and temporalis muscles of both sides simultaneously. There is wasting and weakness of the muscles on side of paralysis.

Sensory function: Test light touch (cotton wool), temperature (cold and hot) and pain (pinprick) on whole face in area of distribution of trigeminal nerve (Fig. 17.12) and compare the two sides.

Corneal reflex: Test corneal sensation by touching with wisp of cotton wool. A blink response should occur bilaterally. In this reflex, afferent route is ophthalmic division of V nerve and efferent route is facial nerve. This test is most sensitive indicator of trigeminal nerve damage.

Jaw jerk: Ask the patient to relax jaw and place finger on the chin and tap with hammer. Slight jerk is normal. But increased jerk is due to bilateral upper neuron lesion.

Facial Nerve (VII)

Observe the patient as he talks and smiles for:

- Eye closure.

- Flattening of nasolabial folds.
- Asymmetric elevation and pulling of one angle of mouth.

Then ask the patient to:

- Wrinkle forehead by looking upwards (*frontalis*).
- Close eyes while examiner attempts to open them (*orbicularis oculi*).
- Show teeth (*orbicularis oris*).
- Puff out the cheeks while examiner presses the cheeks (*buccinator*).

Taste is tested by using salt, sugar and quinine. A small quantity is placed on anterior 2/3rd of tongue.

Auditory Nerve (VIII)

Test the power of hearing by placing the watch near one ear and then compare with other side. If hearing is impaired, examine external auditory canal to exclude wax or infection. Conductive (nerve) deafness is differentiated from perceptive (middle ear) deafness by:

- Weber's test:* Base of vibrating tuning fork is put against vertex and ask the patient to compare sound in two ears. It compares bone conduction on two sides.
- Rinne's test:* Hold the base of vibrating tuning fork against mastoid bone and ask patient if note is heard (bone conduction). Then hold vibrating tuning fork near external meatus and ask patient to hear sound again (air conduction). In conductive deafness, bone conduction is better than air conduction. In nerve deafness, both air and bone conduction are impaired.

Glossopharyngeal Nerve (IX)

Depress patient's tongue and test for sensations with a probe touching the back of pharynx, tonsil and posterior one-third of tongue. Compare sensitivity on both sides.

Vagus Nerve (X)

Ask the patient to open mouth and say 'Ah'. Look for asymmetry of palatal movements. In paralysis, affected half of palate will remain immobile and the uvula moves to the normal side.

Accessory Nerve (XI)

Sternomastoid muscle: Ask the patient to rotate head against resistance. Compare power and muscle bulk on

each side. The left sternomastoid turns the head to the right and vice versa.

Trapezius muscle: Ask the patient to 'shrug' shoulders against resistance and compare power on each side.

Hypoglossal Nerve (XII)

Ask the patient to protrude tongue. In paralysis, the tongue deviates to the paralyzed side. In long standing cases, affected half of tongue becomes atrophic.

The causes and clinical presentation of cranial nerve damage is given in Box 17.15.

TRIGEMINAL NEURALGIA (TIC DOULOUREUX)

Most commonly seen in middle aged or elderly females. It is characterized by intermittent attacks of severe, sharp, stabbing pain affecting second or third division of trigeminal nerve while first (ophthalmic) division is mostly spared. The precipitating factors for pain are:

- Exposure to cold
- Chewing
- Tooth brushing
- Talking
- Touching specific points on face (trigger zones).

The attack of pain lasts for several days or weeks. When the attack settles, patient may remain pain free for many months. Etiology remains unknown in most cases. However, ectatic vascular loops are found to cause compression of trigeminal nerve.

Sometimes trigeminal pain is seen in tumors of cerebello-pontine angle and 8th nerve tumor (acoustic neuroma) but in such cases pain is continuous with associated physical signs of causative lesion.

Investigations

MRI to exclude cerebello-pontine angle lesions.

Treatment

- Drug therapy:* Carbamazepine is effective in pain control in most cases and also helps in confirming the diagnosis. Dose is increased (600-1600 mg/day) till pain is relieved. Side effects are ataxia and drowsiness. When remission occurs, drug treatment can be stopped.
- Operative therapy:* It is indicated when drug therapy fails.

Box 17.15: Cranial nerve damage

<i>Nerve</i>	<i>Cause of damage</i>	<i>Presentation</i>
Olfactory (I)	• Fracture anterior cranial fossa (ethmoid bone)	Impaired sense of smell
Optic (II)	• Rise in intracranial pressure • Glial tumors	Impaired vision
Oculomotor (III)	• Fracture anterior cranial fossa • Cerebral herniation	Fixed dilated pupil, Ptosis, Squint
Trochlear (IV)	• Fracture anterior cranial fossa	Squint
Trigeminal (V)	• Pressure on the nerve	Paralysis of masseter and temporalis muscles, Trigeminal neuralgia.
Abducent (VI)	• Fracture base of skull	Diplopia
Facial (VII)	• Acoustic neuroma • Bell's palsy • Trauma during parotidectomy	Facial palsy
Auditory (VIII)	• Acoustic neuroma • Fracture base of skull	Hearing loss
Glossopharyngeal (IX)	• Fracture base of skull	Loss of gag reflex
Vagus (X)	• Fracture base of skull • Thyroid surgery (recurrent laryngeal nerve damage)	Palatal paralysis Recurrent laryngeal nerve palsy
Accessory (XI)	• Fracture base of skull • Surgery in posterior triangle of neck (Lymph node biopsy, neck dissection)	Sternomastoid paralysis Trapezius paralysis (Drooping shoulder, winging of scapula)
Hypoglossal (XII)	Injury during submandibular gland excision	Deviation of tongue

- Trigeminal ganglion alcohol injection.
- Radiofrequency thermocoagulation of trigeminal ganglion.
- Trigeminal root section.
- Microvascular decompression of trigeminal nerve root in the posterior fossa.

FACIAL NERVE PARALYSIS**Surgical Anatomy**

Facial nerve contains mainly motor fibers supplying muscles of facial expression. Its sensory branch (chorda tympani) carries taste fibers from anterior 2/3rd of the tongue. The muscles in the lower half of face are controlled by contralateral hemisphere while those in the upper face receive control from both hemispheres (bilateral representation). Hence, a lower motor neuron lesion paralyses all facial muscles on that side while an upper motor neuron lesion (supranuclear) paralyses only muscles in the lower half of the face on opposite side.

Causes of Facial Nerve Paralysis*Upper motor neuron lesions*

- Vascular (cerebrovascular accidents)
- Tumors
- Infection (meningitis)

Lower motor neuron lesions

- Fracture base of skull
- Malignant parotid tumor (see Fig. 15.10)
- Parotid gland surgery (Fig. 17.13)
- Otitis media.
- Bell's palsy
- Facial trauma
- Herpes zoster (Ramsay-Hunt syndrome)

Clinical Features

On affected side:

- Forehead does not wrinkle.
- Eye fails to close and on attempting, eyeball rolls upwards and outwards (Bell's phenomenon).



Fig. 17.13: Right facial nerve palsy following parotidectomy

- On showing teeth, nasolabial fold is flattened and angle of mouth droops.
- Drooling of saliva through angle of mouth.
- Taste impairment in anterior 2/3rd of tongue.

However, in upper motor lesion, there is preservation of eye closure and forehead wrinkling due to bilateral representation.

Clinical symptoms of facial nerve damage vary based on level of injury (Fig. 17.14).

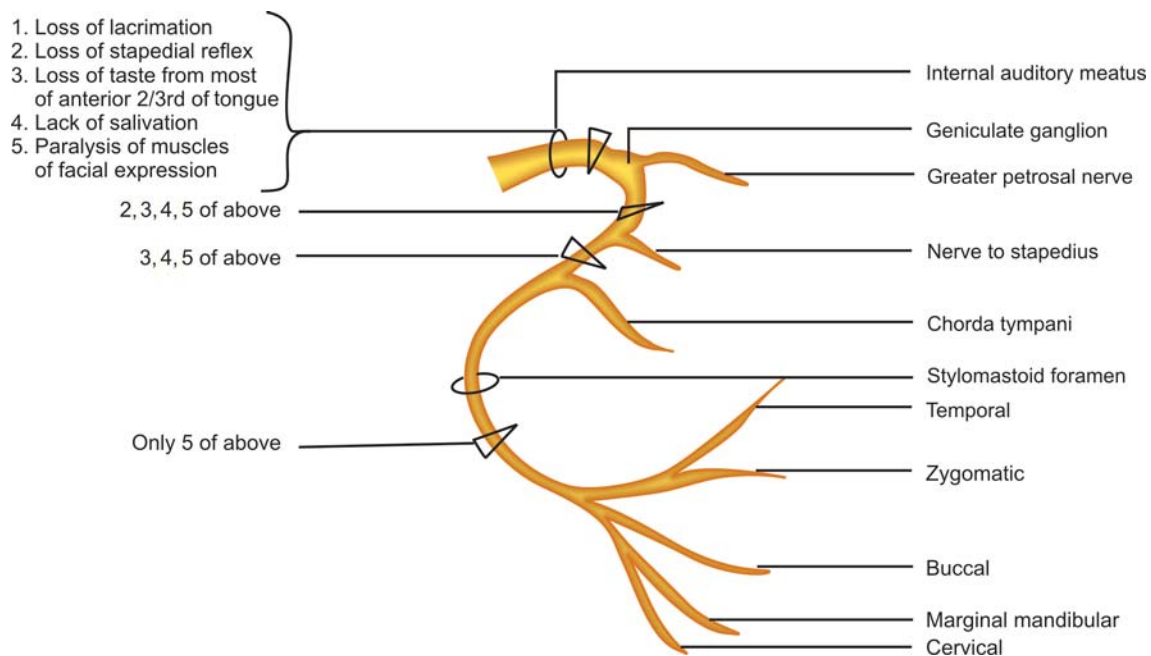


Fig. 17.14: Effects produced by facial nerve damage at various levels

Bell's Palsy

- It is characterized by acute paralysis of face related to inflammation and swelling of the facial nerve within the facial canal.
- It is mostly unilateral, rarely bilateral.
- It may occur repetitively.
- Its exact etiology is not known but may be associated with viral infection.
- In some cases, there is positive family history.

Investigations

CT/MRI of head is done if some intracranial lesion is suspected.

Treatment

Medical management

- In acute stage of Bell's palsy, prednisolone is given in high doses 40-60 mg/day that is gradually tapered over 7-10 days.
- Acyclovir 800 mg 5 times a day is given orally in viral infection (Bell's palsy, Herpes zoster).
- Eye shielding to prevent corneal abrasions.
- Methyl cellulose eye drops (artificial tears) to prevent dryness.

Surgical management:

- Tarsorrhaphy to prevent exposure keratitis in eye.
- If facial nerve injury is identified during surgery (e.g. parotidectomy), the nerve is repaired primarily. In case there is gap between two cut ends of the facial nerve, a sural or greater auricular nerve graft can be put in.
- If patient presents later when direct repair is not possible, a nerve transfer of hypoglossal to the facial nerve can be done.
- Plastic surgical procedures to improve resting state of the face:
 - ☐ Face lift operation.
 - ☐ Insertion of slings of fascia lata.
 - ☐ Transplanting the insertion of temporalis muscle (with its nerve supply intact) from mandible to the cheek to support corner of mouth.

Plastic operation should be delayed for 1-1½ years after onset of paralysis when all hopes of spontaneous recovery of facial nerve are lost.

18

Chapter

Gangrene and Diseases of Arterial System

Sanjay Marwah

GANGRENE

Gangrene is defined as macroscopic death of the tissues with superadded putrefaction. Thus, formation of gangrene involves tissue necrosis followed by bacterial infection leading to decay and putrefaction of the dead tissues.

The organs commonly affected by gangrene are:

- Distal parts of upper and lower limbs.
- Abdominal viscera (appendix, intestines, gall-bladder).

Causes of Gangrene

1. *Arterial obstruction (ABCDE)*
 - A. Arterial thrombosis (Atherosclerosis)
 - B. Buerger's disease
 - C. Cervical rib
 - D. Drugs (ergotamine, thiopentone)
 - E. Embolism
 - F. Raynaud's disease
2. *Venous obstruction:* Deep vein thrombosis.
3. *Traumatic causes:*
 - a. *Direct* arterial injury, e.g. in crush injury, pressure sores.
 - b. *Indirect* arterial injury in which vessel is injured at some distance from the site of gangrene, e.g. fractured bone fragment pressing on an adjoining artery.
4. *Infective causes:* Boil, carbuncle, cancrum oris, gas gangrene, Fournier's gangrene.
5. *Diabetic gangrene:* Angiopathy, neuropathy and infection act together in causation of gangrene.
6. *Physical causes:*
 - Heat: Burns and scalds
 - Cold: Frostbite, trench foot
- Chemicals

- Irradiation
- Electricity

Clinical Features

The gangrenous part has:

- No arterial pulsations, venous return and capillary filling
- Cold to touch
- No sensations
- No functions
- Color changes with passage of time. Initially it is dusky gray and gradually changes to dark brown, greenish and finally black in color. The color changes are due to red cell destruction and formation of iron sulphide (Box 18.1A).

Clinical Types

There are two types:

- Dry gangrene
- Moist gangrene

Dry gangrene

- There is gradual occlusion of arterial supply while the venous return remains unimpaired.
- It is typically seen in arterial thrombosis due to senile atherosclerosis, Buerger's disease.
- The gangrenous part appears 'mummified' and is dry, dark and wrinkled.
- A definite line of demarcation appears between the living and dead tissue and there is no infection. It is called as *separation by aseptic ulceration* (Fig. 18.1B).

Box 18.1A: Gangrene—clinical findings

- Loss of pulsations
- Loss of temperature
- Loss of function
- Loss of sensations
- Loss of color



Fig. 18.1A: Dry gangrene of tip of middle finger (Atherosclerotic); line of demarcation between living and dead tissue is visible



Fig. 18.2: Moist gangrene of leg having swollen and discolored skin with blebs. There is no line of demarcation



Fig. 18.1B: Dry gangrene of scalp (Postburn)



Fig. 18.3: Moist gangrene scalp (Traumatic)

- If there is underlying bone in gangrenous tissue, e.g. finger, the separation takes longer and final appearance of stump after separation is conical since bone is more vascular than covering skin and soft tissues (Fig. 18.1A).

Moist gangrene

- There is sudden occlusion of arterial supply along with blockage of venous return.
- Common causes of moist gangrene are embolism, diabetic gangrene and gas gangrene.
- The gangrenous part appears swollen, discolored and skin is raised into blebs containing foul smelling fluid.
- Crepitus may be palpable along with foul smelling odor due to gas forming organisms.

- Due to edema and infection, formation of line of demarcation is delayed and final line of demarcation appears at a much higher level. It is called as *separation by septic ulceration* (Figs 18.2 and 18.3).

Spread of Gangrene

- When the line of demarcation between living and gangrenous tissue is vague, it indicates that the arterial supply of living tissue is compromised.
- If blood supply to living tissues is not improved, then there is sudden appearance of dark patches in adjoining healthy area and gangrene spreads to proximal part (phenomenon of 'die back').
- It mostly happens in wet gangrene where infection is present and collateral circulation fails to develop.

Box 18.1B: Dry gangrene vs moist gangrene

	Dry gangrene	Moist gangrene
Mechanism	Gradual arterial occlusion	Sudden arterial occlusion
Etiology	Senile atherosclerosis	Infective
Clinical features	• Dry • Mummified • Nonsmelling • Line of demarcation seen	• Wet • Edematous • Foul smelling • No line of demarcation
Treatment	Conservative amputation	Major amputation (life saving)

- So every attempt should be made to convert a wet gangrene into a dry gangrene by regular dressings, antibiotics and treatment of underlying cause (e.g. diabetes). It helps in formation of line of demarcation and prevents spread of gangrene.
- Comparison between dry and moist gangrene is summarized in Box 18.1B.

Treatment of Gangrene

General measures

- Improvement in wound healing by nutrition.
- Improvement in tissue oxygenation by treatment of:
 - ☐ Heart failure.
 - ☐ Arrhythmias.
 - ☐ Anemia.
- Control of diabetes mellitus.
- Control of pain with analgesics (preferably non-narcotic analgesics).
- Control of infection with antibiotics.

Local treatment

- Care of the affected part:
 - ☐ Keep the part dry by exposure and use of fan.
 - ☐ Keep the part elevated for decreasing edema and pain.
 - ☐ Do not heat the part.
 - ☐ Protect the local pressure areas on heel, malleoli, back especially in cases of gangrene due to neurological causes (paraplegia, diabetic neuropathy, etc.). A foam padding or water bed may prevent pressure sores.
- Minor surgical toilet in form of drainage of pus pockets, debridement of slough and removal of crust should be done regularly. The aim is to convert a wet gangrene into a dry one.

Surgical treatment

- **Direct arterial surgery:** It has role in further progress of ischemia in proximal parts. The details are discussed under individual causes of gangrene.

- **Sympathectomy**

- **Amputation:** It has two aims:

- Life saving:** In cases of badly crushed limbs where moist gangrene is spreading rapidly and causing systemic sepsis, e.g. gas gangrene. Here urgent amputation is required to save the life of the patient.
- Limb saving:** In cases of dry gangrene affecting limbs, aim is to salvage as much limb as possible. So, with conservative treatment or surgical treatment (arterial repair, sympathectomy) blood supply of the limb is improved. It helps in formation of line of demarcation and a major amputation may be avoided.

INDIVIDUAL CAUSES OF GANGRENE

ATHEROSCLEROTIC ARTERIAL THROMBOSIS

- Atherosclerosis is a degenerative condition chiefly affecting large and medium sized arteries.
- The arterial thrombosis involves 'intima' and leads to blockade of vessel lumen.
- Abdominal aorta, iliac and femoral arteries are commonly involved.
- Involvement of upper extremity vessels is very rare.
- The arteries of heart and brain are frequently affected leading to myocardial infarction and stroke respectively.
- The involvement of lower limb vessels present as *chronic limb ischemia*.
- The severity of symptoms depends upon size of vessel occluded and presence of collateral vessels. A useful practical classification is shown in Box 18.2A.

Symptoms

- **Intermittent Claudication (To limp)**
 - ☐ Crampy pain felt in leg muscles on walking.
 - ☐ The site and extent of pain is related to extent of the disease (Box 18.2B).



Fig. 18.4A: Buerger's posture test—pallor on limb elevation

Box 18.2A: Fontaine classification of lower limb ischemia

Stage I	Asymptomatic
Stage II	Intermittent claudication
Stage III	Rest pain
Stage IV	Ulceration with or without gangrene

Box 18.2B: Symptoms based on extent of disease

Femoropopliteal disease	Calf Claudication
Ileofemoral disease	Thigh Claudication
Aortoiliac disease	Buttock Claudication + impotence in males (Leriche's syndrome)

- ☐ The pain increased steadily and patient is compelled to stop.
- ☐ The pain is relieved on taking rest.
- ☐ The distance walked is called 'claudication distance'.
- ☐ It means that collateral circulation is providing enough blood for the relevant muscles at rest but not during activity.
- **Rest pain**
 - ☐ Severe pain felt in the foot even at rest.
 - ☐ Cause of pain is ischemia of nerves (Cry of dying nerves).
 - ☐ Pain is worse at night when limb becomes warm under bed clothes that increases the oxygen requirements.



Fig. 18.4B: Buerger's posture test—congestion on limb dependence

- ☐ Pain is somewhat relieved by hanging the leg or sleeping in chair because dependency increases the blood flow.
- There is *coldness*, *numbness* and *paresthesia* in the affected limb.
- **Color changes:** These can be demonstrated with *Buerger's posture test*. On limb elevation, it becomes pale and in dependent position, it becomes cyanosed. This sequence indicates that a major artery is occluded (Figs 18.4A and B).
- **Ulceration:** Painful superficial ulcers are seen around malleoli, on dorsum of foot, on shin or in between toes.
- **Pre-gangrene:** The combination of rest pain, color changes, coldness, numbness, paresthesia with or without ulceration is called as stage of pre-gangrene.
- **Gangrene:** It is usually dry type because chronic limb ischemia gives sufficient time for collaterals to develop and it affects the toes and later it may extend proximally to involve variable part of leg (Fig. 18.5).

Signs

- **Trophic changes** in the limb are signs of chronic ischemia (Box 18.2C). These are loss of hair, brittle and opaque nails, skin atrophy, loss of subcutaneous fat, muscle wasting, bone wasting. Even trivial trauma (improper shoes, pairing of nails) can result in increased metabolic demand in such a limb. It leads to thrombosis of collateral circulation causing gangrene.

Box 18.2C: Trophic changes

- Signs of chronic limb ischemia.
- Compare affected limb with healthy limb.
- Findings are
 - ☐ Thin limb.
 - ☐ Loss of hair.
 - ☐ Brittle nails.
 - ☐ Skin atrophy.
 - ☐ Loss of subcutaneous fat.
 - ☐ Muscle wasting.
 - ☐ Bone wasting



Fig. 18.5: Atherosclerotic dry gangrene of the toes

- **Arterial pulsations:** These are usually absent below the site of arterial obstruction and diminished in presence of good collateral circulation. Diminished pulses can be appreciated by comparing it with other limb pulses provided that is normal. The method of feeling various pulses is shown in Box 18.3.
- **Venous refilling:** The affected limb is elevated for thirty seconds and then laid down on bed. Normal refilling occurs within seconds. Decreased venous refilling is a sign of severe arterial insufficiency. Venous refilling can also be examined by *Harvey's*



Fig. 18.6: Palpation of dorsalis pedis artery

Box 18.3: Method of feeling the pulses

Lower limb pulses

- Dorsalis pedis artery (Continuation of ant. tibial artery)
 - Felt in proximal part of groove between first and second metatarsal bones (Fig. 18.6) (Congenitally absent in 10% cases).
- Posterior tibial artery (Continuation of popliteal artery)
 - Felt halfway between back of medial malleolus and medial border of tendo-Achilles (Fig. 18.7).
- Popliteal artery (Continuation of femoral artery)
 - Flex the knee while patient is supine or prone. Start palpating in popliteal fossa from medial to lateral side. The artery is felt in the middle part of fossa against lower end of femur (Fig. 18.8).
- Femoral artery (Continuation of external iliac artery)
 - Palpate below the inguinal ligament midway between anterior superior iliac spine and symphysis pubis (Fig. 18.9).

Head and Neck pulses

- Superficial temporal artery (Terminal branch of ext. carotid artery)
 - Felt in front of tragus against zygoma.
- Common carotid artery (Origin: On left side—arch of aorta, on right side—brachiocephalic artery)
 - Felt in carotid triangle at level of Adam's apple (upper border of thyroid cartilage)
- Subclavian artery (Origin: On left side—arch of aorta, on right side—brachiocephalic artery)
 - Felt in supraclavicular fossa against first rib.



Fig. 18.7: Palpation of posterior tibial artery



Fig. 18.8: Palpation of popliteal artery



Fig. 18.9: Palpation of femoral artery in groin



Fig. 18.10A: Harvey's sign—emptying of a venous segment with two fingers



Fig. 18.10B: Harvey's sign—venous refilling on releasing distal finger

sign (Figs 18.10A and B). Two index fingers are used to empty a segment of limb vein. The release of distal finger allows venous refilling.

- *Capillary refilling:* Pressure is applied on tip of terminal pulp space for a few seconds and then released. Slow disappearance of blanching is a sign of severe arterial insufficiency.
- *Examination of heart:* For heart sounds and any murmurs.
- *Arterial bruit:* Auscultation of major vessels is done to listen for systolic murmurs due to arterial stenosis.
 - ☐ Subclavian artery in supraclavicular fossa.
 - ☐ Carotid artery in neck behind angle of mandible.
 - ☐ Abdominal aorta in supraumbilical region.

- ☐ Femoral artery in groin.
- ☐ Popliteal artery in popliteal fossa.

Continuous machinery murmur is a sign of 'arterio-venous fistula'.

Investigations

- **General investigations**
 - ☐ Full blood count including ESR and platelets to look for anemia and any hematological disorder.
 - ☐ Blood sugar for diabetes.
 - ☐ Serum cholesterol and lipid profile to look for abnormal lipid metabolism (atherosclerosis).
 - ☐ Blood urea and serum creatinine for renal functions.
 - ☐ ECG to look for coronary artery disease.
 - ☐ Echocardiography and treadmill test may be required in case of underlying heart disease.
- **Doppler ultrasound blood flow:** An ultrasound beam is made to strike the moving blood in a vessel and it is reflected back. It leads to shift in ultrasound frequency (the Doppler effect) that is picked up as audio signals. It indicates velocity of blood flow in the vessel. It can be used as a sensitive stethoscope with sphygmomanometer to assess arterial blood pressure even at sites where vessels are not palpable. A simple and valid test for lower limb ischemia is by measuring ankle/arm blood pressure ratio (Pressure index). Normally pressure index is 1 or higher. In claudication it is between 1 and 0.5. In severe ischemia, it is <0.5. Fall in pressure index after exercise indicates occult arterial stenosis.
- **Color Duplex imaging:** B-mode ultrasound is used to provide image of vessel. It is combined with Doppler ultrasound and then analyzed by a computer. There is color coding that indicates change in direction and velocity of blood flow. Area of arterial stenosis is picked up as "high flow" area. Its advantage is that it is non-invasive test and accuracy is equivalent to angiography in picking up arterial stenosis.
- **Angiography (Arteriography):** It is an invasive investigation and has its own complications (Box 18.4). Hence, it is performed only when surgical intervention has been decided in a case. It is done as follows:

Seldinger's technique: Femoral artery is punctured with Seldinger's needle. A guide wire is passed through the needle into aorta and needle is removed. A catheter is passed over guide-wire and the wire is

removed. The radiopaque dye is injected into arterial tree and radiographs are taken. It outlines the site and length of arterial obstruction as well as collateral vessels.

Box 18.4: Complications of angiography

- | | |
|---------------|-----------------------|
| • Anaphylaxis | • Paraplegia |
| • Hematoma | • Renal failure |
| • Thrombosis | • Arterial dissection |

- **Digital subtraction angiography (DSA):** A computer system is used to digitalize the angiographic findings. The computer subtracts the extra background findings and outlines the vessels only thus providing greater clarity. It can be performed by arterial as well as venous injection of contrast material.
 - **CT angiography:** With availability of multislice CT scan, it can be used to image vessels. It can cover thorax, abdomen and pelvis in a single breath-hold. It is relatively noninvasive and can be performed on OPD basis. It provides three-dimensional view of vascular anatomy. Another advantage is that it visualizes vessel wall, thrombus within the lumen and structures around the vessel. However, it requires ionizing contrast as well as radiation as in conventional angiography.
 - **Magnetic resonance angiography:** It provides imaging without need of ionizing radiation or direct arterial puncture. It has better visualization of patent distal vessels when flow is minimal. Also it visualizes vessels in three dimensions. It is a costly investigation and cannot be performed in presence of metal objects (metal implants, pacemakers, etc.).
- Box 18.5 compares various recent imaging techniques.

Management

- The list of risk factors for arterial diseases and their management is shown in Box 18.6.
- **Pain control:**
 - ☐ Patient of intermittent claudication is afraid of walking. Once explained that walking is useful, patients are able to improve their claudication distance due to development of collateral circulation. Raising of shoe heel by 1 cm reduces the work load of calf muscles and improves claudication distance.

Box 18.5: Comparison of recent imaging techniques

	DSA	CT angio- graphy	MR angio- graphy
Uses ionizing radiation	Yes	Yes	No
Uses ionizing contrast	Yes	Yes	No
Invasive	Yes	No	No
Images extravascular structures	No	Yes	Yes
Three-dimensional image	No	Yes	Yes
Contraindicated in presence of metals	No	No	Yes

- ☐ Patients of rest pain require analgesics. To begin with simple analgesics like paracetamol or aspirin should be given. Narcotic analgesics (Tramadol, Pethidine) should only be used as a last resort.
- ☐ Rest pain can also be relieved by:
 - a. Buerger's position: Elevation of head end of the bed.
 - b. Buerger's exercises: Alternate elevation and dependency of the limb for 2 minutes each.
- **Patient education:** It is regarding protection of affected limb from any form of trauma. It includes:
 - ☐ extremes of temperature (heat or cold)
 - ☐ trimming of nails, corns, etc.
 - ☐ Ill-fitting shoes
 - ☐ Skin infections
 - ☐ Foot care (skin lubrication with moisturizer, lamb's wool between toes)
- **Vasodilators:**
 - ☐ They have doubtful role in chronic limb ischemia.

Box 18.6: Risk factors and their management

Hypertension	Antihypertensive drugs
Diabetes mellitus	Dietary control
	Oral hypoglycemics
	Insulin
Obesity	Dietary control, exercise
Sedentary lifestyle	Exercise
Smoking	Smoking cessation (counseling, nicotine replacement)
(Tobacco is potent vasoconstrictor)	
Hypercholesterolemia	Dietary manipulations
	Statin therapy
Vascular disease	Antiplatelet drugs
	(Disprin, clopidogril)

- ☐ There may be some improvement in pain and superficial ulcers may heal.
- ☐ The drugs are xanthinol nicotinate (complamina), pentoxifylline (Trental), calcium channel blocker (nifedipine).

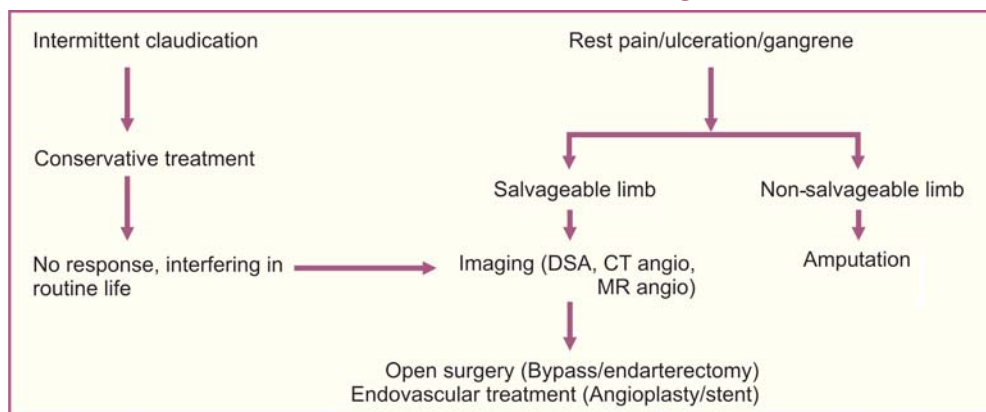
Surgery for Chronic Lower Limb Ischemia

Indications

- **Intermittent claudication:** It is mostly treated conservatively and surgery is not performed. However, if it is not responding to medical management and is interfering with routine life of the patient, surgery may be performed.
- **Rest pain, ischemic ulceration and pregangrene** are absolute indications for surgery.

Outline for management of chronic lower limb ischemia is given in Box 18.7.

Once surgery is decided, radiological imaging (DSA/CT angio/MR angio) is done to plan the type of surgical procedure.

Box 18.7: Chronic lower limb ischemia: Management outline

Various surgical procedures available are:

Open Surgery

1. Thromboendarterectomy

- It is performed when disease is affecting large arteries (aorta, iliac artery) and only a short segment of artery is involved.
- An arteriotomy is performed at site of obstruction and diseased intima, atheromatous plaque and thrombus are removed.
- The arteriotomy is closed primarily. In case, there is fear of luminal narrowing, a vein patch can be used to close the arteriotomy wound.

2. Bypass Graft

- It is performed when large and medium sized vessels (up to popliteal artery) are involved.
- It has no role in occlusion of distal small sized vessels.
- It is useful in bypassing multiple sites of occlusion.
- Material used for bypass graft can be:
 - prosthetic materials like Dacron, Polytetra fluoroethylene (PTFE). It is used in aortoiliac block.
 - Patient's own long saphenous vein of the same limb. Since long saphenous vein has valves which do not allow blood flow from proximal to distal side of the limb so either reverse long saphenous vein is used or *in situ* long saphenous vein is used after valve disruption. It is used in femoropopliteal block.
- In patients having severe ischemia and unfit for major surgery, *extra-anatomical bypass grafting can be done*.
- The types of bypass graft are described in Box 18.8.

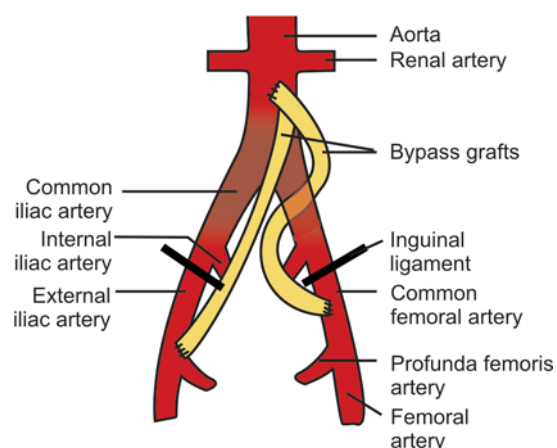


Fig. 18.11: Aortofemoral bypass graft

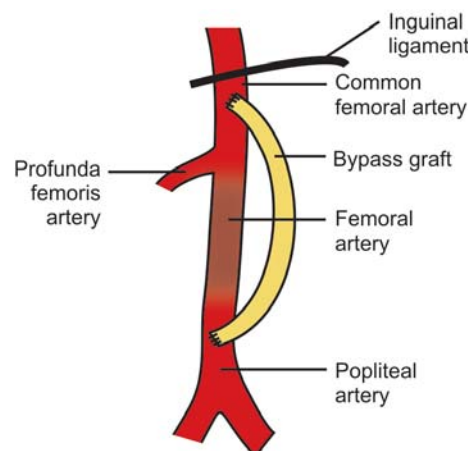


Fig. 18.12: Femoropopliteal bypass graft

Box 18.8: Bypass grafts

Disease site	Type of Bypass	Material used
Aortoiliac disease	Aortofemoral bypass graft (Fig. 18.11) (between infrarenal aorta and common femoral artery).	Dacron, PTFE
Iliofemoral disease	Iliofemoral bypass graft (between common iliac and common femoral artery)	Dacron, PTFE
Femoropopliteal disease	Femoropopliteal bypass graft (Fig. 18.12) (between femoral and popliteal artery)	Long saphenous vein (reverse or <i>in situ</i>)
Aortoiliac disease (Patient unfit for major surgery)	Extra-anatomical bypass graft - Axillofemoral graft (between axillary artery and femoral artery of same side) - Femorofemoral crossover graft (Between ipsilateral femoral artery and contralateral femoral artery)	Dacron, PTFE

3. Profundaplasty

- It is performed when there is stenosis of origin of profunda femoris artery.
- An incision is made into common femoral artery and carried down into the profunda femoris artery so as to divide the stenotic area.
- The arteriotomy is then closed with a vein patch to widen the narrow segment.

Endovascular Treatment

Percutaneous Transluminal Angioplasty (PTA)

- It is performed when only a short segment of artery is occluded.
- It has major success in dilating iliac artery occlusion.
- It can be used up to legs for dilating vessels.
- It is performed in radiology department under fluoroscopic control using local anesthesia.
- A guide-wire is passed across stenosis.
- A balloon catheter is then inserted over the guide-wire and inflated for one minute in the area of stenosis. The inflation and deflation is done twice before removing catheter after final deflation.
- Laser can be combined with angioplasty to drill hole in the narrow segment.
- After balloon dilation, a self-expandable *metal stent* may be placed at site of stenosis to maintain patency.

EMBOLISM

- Embolus is a body that is foreign to the bloodstream (usually a clot) and gets lodged in an artery causing sudden arterial occlusion.
- The *sources* of blood clot are:
 - Mural thrombus in heart (due to myocardial infarction, arrhythmia, mitral stenosis)
 - Aneurysms of thoracic/abdominal aorta
- The embolus may lodge in any organ leading to ischemic necrosis and infarction, e.g. brain, lungs, kidneys, retina.
- In lower limbs, embolus may block a major vessel leading to ischemia and gangrene.

Clinical Features

- The symptoms are sudden in onset without any previous history of intermittent claudication.
- The features can be remembered by '5P':

Pain:	Sudden, severe, excruciating pain.
-------	------------------------------------

- | | |
|----------------|---|
| Pallor: | The limb is dead white with bluish discoloration. |
| Paralysis: | Patient is unable to move the limb. |
| Pulselessness: | No pulses felt distal to obstruction. |

- | | |
|---------------------------|---------------------------|
| Paresthesia (Anesthesia): | Limb sensations are lost. |
| Poikilothermia: | Cold limb. |

- Embolic occlusion needs to be differentiated from thrombotic occlusion due to atherosclerotic disease (Box 18.9).
- Tissue ischemia usually develops one joint level below the segment of occluded artery, e.g. femoral artery occlusion will produce ischemia distal to knee joint.
- Embolic occlusion is an emergency requiring urgent surgical intervention.
- If left untreated, ischemic necrosis begins in 6 to 8 hrs. and gangrene can develop very rapidly.
- *Diagnosis:* In majority of cases, arteriography is needed for precise location of arterial occlusion.
- *Treatment:*
 - I/V heparin infusion (5000-10000 units) should be started early to prevent extension of clot.
 - I/V infusion of fibrinolytic agents (urokinase, streptokinase) to lyse the clot without doing surgery. It is more effective in acute thrombosis than in embolism. There is high-risk of hemorrhage, infection and anaphylactic reaction, Heparin should not be used along with fibrinolysis.
 - *Emergency embolectomy* is done under local or general anesthesia. Arteriotomy is done at site of clot, clot removed and arteriotomy closed. Distally placed embolus remote from arteriotomy can be removed for using a Fogarty balloon catheter (Fig. 18.13).

BUERGER'S DISEASE

- It is occlusive arteritis affecting medium and small sized arteries, also known as *thromboangiitis obliterans (TAO)*.
- The vessels usually involved are dorsalis pedis, posterior tibial, popliteal and radial arteries.
- It is a disease of chronic smokers affecting young males (<30 yrs of age).
- There is segmental local inflammation in the walls of arteries and veins leading to thrombosis.

Box 18.9: Differences between embolism and thrombosis

	<i>Embolism</i>	<i>Thrombosis</i>
Past history of claudication	—	+
Source of embolus, e.g. recent MI, arrhythmia	+	—
Symptoms	Sudden onset	Long standing
Limb color	Pale, dead white	Congested
Paralysis	+	—
Anesthesia	+	—
Trophic changes	—	+
Angiography	No collaterals	Collaterals +
Treatment	Emergency intervention. Removal of embolus.	Not an emergency. Removal of underlying lesion.

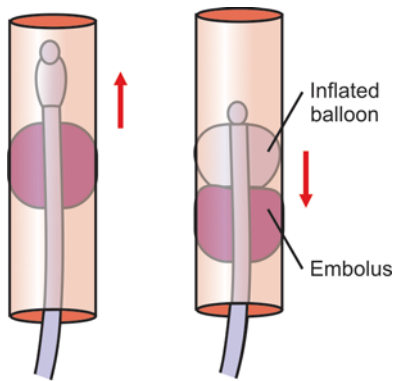


Fig. 18.13: Embolectomy—balloon inflation distal to embolus and embolus extraction

- Involvement of upper extremity vessels is not unusual.
- The disease has classical triad of:
 - i. Chronic limb ischemia (intermittent claudication, ulceration, rest pain, gangrene).
 - ii. Thrombophlebitis affecting superficial or deep veins.
 - iii. *Raynaud's syndrome*: On exposure to cold, the digits become painful along with color changes in sequence from pallor to cyanosis and then finally red in color.
- The patient may have one, two or all the three manifestations.
- Gangrene of toes and fingers is common and progressive (Fig. 18.14).
- One of the important differential diagnosis is presenile atherosclerosis (Box 18.10).



Fig. 18.14: Dry gangrene toes—Buerger's disease

Investigations

General investigations: Same as for atherosclerotic thrombosis (described above).

Color duplex imaging: It shows occlusion of medium and small sized vessels. Large vessels are normal.

Angiography: It shows:

- Occlusion of distal vessels.
- Normal proximal arteries (no atherosclerosis).
- *Segmental occlusive lesions*: Diseased arterial segments interspersed with normal appearing segments.
- Formation of 'cork screw' collaterals around area of occlusion.

Box 18.10: Differences between Buerger's disease and presenile atherosclerosis

<i>Buerger's disease</i>	<i>Presenile atherosclerosis</i>
Inflammatory arteritis leading to arterial occlusion	Degenerative arterial disease causing arterial occlusion.
The disease involves full thickness of arterial wall (panarteritis)	The disease involves intima leading to formation of 'atheroma' and thrombus
Disease involves medium and small sized vessels	Disease involves medium and large sized vessels.
Upper limb vessels—involved	Upper limb vessels—not involved
Veins—involved	Veins—not involved
Raynaud's syndrome—present	Raynaud's syndrome—not seen.
Heart and brain vessel involvement—absent	Heart and brain vessels—involved
It is common in young males who are chronic smokers with poor socioeconomic background	It is commonly seen in middle or elderly patients (male or female) who are rich, obese and have family history of the disease.
Angiography: It shows obliterated distal vessels with typical 'cork screw' collateral vessels.	Angiography shows site of thrombus in medium or large vessel, distal run off (blood flow in vessels distal to obliteration) and collaterals.
Direct arterial surgery (bypass, thromboendarterectomy) is not useful in improving limb perfusion.	Direct arterial surgery useful in improving limb perfusion.
Lumbar sympathectomy, Omentopaxy: Have some role in cutaneous vasodilatation and may heal superficial ulcers.	No role.
Amputation: Usually minor	Major, if required.

Treatment

- Abstinence from smoking is most important. It will stop the progress of disease. However, it will not reverse the already established arterial occlusion. The patient should be told clearly that:
“Either you can have your cigarettes or you can have your limbs. You can not have both things together!”
- Antibiotics for cellulitis, NSAIDs for superficial phlebitis.
- Pain control, patient education, vasodilators (See management of atherosclerosis).
- *Lumbar sympathectomy*:
 - ☐ It causes cutaneous vasodilatation and may help in relieving rest pain and healing ulcers.
 - ☐ It is not effective in intermittent claudication.
 - ☐ Indications of lumbar sympathectomy are given in Box 18.11.
 - ☐ It can be performed in two ways:
- a. *Chemical Sympathectomy*
 - i. Patient in sitting position.
 - ii. 15 cm long needle is inserted in paravertebral region under fluoroscopic control to reach sympathetic trunk.

- iii. 5 ml phenol in water is injected besides bodies of second, third and fourth lumbar vertebrae.
- iv. Care is taken to avoid injury to aorta and vena cava (aspirate the syringe to exclude presence of blood before injecting the drug).
- v. Patient feels warm feet immediately after injection.

Box 18.11: Lumbar sympathectomy: Indications

- Causalgia (Post-traumatic pain syndrome)
- Vasospastic disorders (Raynaud's disease, Frostbite)
- Buerger's disease
- Hyperhidrosis

b. Surgical Sympathectomy

Through transverse lumbar incision, lumbar sympathetic trunk is exposed in paravertebral space. Second, third and fourth lumbar ganglia are removed. If bilateral lumbar sympathectomy is done in a young male, care should be taken to preserve first lumbar ganglion at least on one side to preserve ejaculatory function.

Laparoscopic lumbar sympathectomy helps in early recovery.

- **Omentopaxy:**
 - ☐ It may be tried in an attempt to create neovascularization of the ischemic limb.
 - ☐ Its results are unpredictable and it has doubtful role in managing chronic limb ischemia.
 - ☐ Laparotomy is done. Greater omentum is mobilized based on one of the epiploic arteries, brought out of laparotomy wound, tunneled in a subcutaneous plane and taken up to below knee level in the calf or even up to ankle level.
 - ☐ It may help in relieving rest pain and may heal cutaneous ulcers.
- **Amputation:**
The patient usually develops dry gangrene due to chronic ischemia requiring amputation. Based on the level of amputation, various types are:
 - ☐ Toe amputation—for gangrene of the toe.
 - ☐ Transmetatarsal amputation—for gangrene of multiple toes extending up to forefoot.
 - ☐ Below knee amputation—for cases of severe rest pain and gangrene foot.

RAYNAUD'S DISEASE

- Primary vasospastic disorder usually seen in young females.
- Idiopathic: No identifiable underlying cause.
- Digits of upper limbs are more commonly affected than toes of lower limbs.
- There is abnormal sensitivity of arterioles to the cold exposure.
- The peripheral pulses are normal.
- **Raynaud's phenomenon:** A typical vasospastic attack occurring in response to cold exposure leads to sequential color change of skin starting from pallor, then cyanosis and finally rubor.

Pathophysiology

- Initial *pallor* is due to intense arteriolar spasm leading to blanching of finger tips.
- *Cyanosis* occurs because static blood in capillaries becomes desaturated (Fig. 18.15).
- *Rubor* is because of postischemic vasodilatation leading to increased blood flow.
- The color changes are accompanied with burning pain in the digits.
- Recurrent attacks may lead to gangrenous patches on fingertips (due to superficial necrosis).



Fig. 18.15: Cyanosed fingers in Raynaud's phenomenon

Treatment

- The disease cannot be cured. However, simple measures like “protection from cold” can reduce the frequency and duration of attacks.
- Patient education regarding care of the hands to prevent infection of nailbeds and digits.
- Drugs to relieve vasospasm:
 - ☐ Calcium channel blockers—nifedipine.
 - ☐ α_1 adrenergic blockers—prazosin, terazosin, doxazosin.
- **Cervico-dorsal sympathectomy:**
 - ☐ It is rarely indicated and benefits are short lived.
 - ☐ It may be effective in some patients with severe ischemia and pain in fingertips.
 - ☐ It has high relapse rate due to regeneration of nerve fibers.
 - ☐ It helps in healing ulcers and improving ischemic pain.
 - ☐ It can be done through supraclavicular or axillary route.
 - ☐ Now it can be done through thoracoscopy that reduces postoperative morbidity.
 - ☐ Sympathetic trunk is removed from lower half of stellate ganglion to just below 3rd thoracic ganglion.
 - ☐ Upper half of stellate ganglion is preserved to prevent Horner's syndrome.

Complications

- Pneumothorax

- Chylothorax (thoracic duct injury)
- Horner's syndrome
- Winging scapula (injury of nerve to serratus anterior)
- Phrenic nerve injury

RAYNAUD'S SYNDROME

- Raynaud's phenomenon due to presence of underlying abnormality.
- The causes are:
Atherosclerosis
Thoracic outlet syndrome
Carpel tunnel syndrome
Buerger's disease
Collagen disease (SLE, rheumatoid arthritis)
Occupational (use of vibrating tools, drills)
Drugs (vasopressors, ergot)
Malignancy (Leukemia, multiple myeloma)
- The clinical features are much more aggressive.
- It needs to be differentiated from primary Raynaud's disease (Box 18.12).

Treatment

- Treat the underlying cause.
- Avoid vibrating tools.
- Drugs—steroids, nifedipine.
- Sympathectomy—results are disappointing.

Box 18.12: Distinguishing features between Raynaud's disease and Raynaud's syndrome

Raynaud's disease	Raynaud's syndrome
Idiopathic	Secondary to underlying disease
Usually affects young females	Affects both males and females (any age)
Involves multiple digits	Involves one digit
Pulses are normal	Pulses are weak
No skin abnormalities	Skin lesion (in SLE)
Lab studies—normal	Lab studies—underlying disease

CERVICAL RIB AND THORACIC OUTLET SYNDROME

At root of the neck, various abnormalities may compress the brachial plexus, subclavian artery or subclavian vein near first rib and clavicle to produce symptoms (Box 18.13).

Box 18.13: Causes of thoracic outlet syndrome

- Cervical rib
- Scalenus anticus syndrome
- Costoclavicular syndrome
- Hyperabduction syndrome

Cervical Rib

- It is an extra rib arising from seventh cervical vertebra.
- It is more frequently seen on right side.
- Females are affected in ratio of 2 : 1 to males.
- Paradoxically, cervical rib seen on X-ray is asymptomatic whereas in symptomatic patients, X-ray cervical spine does not show any cervical rib.

Pathophysiology

Normally, brachial plexus and subclavian artery have a smooth curve over upper surface of first rib (Fig. 18.16). In presence of seventh cervical rib, base of the triangle is raised by height of one vertebra. So curve taken by nerve and artery is angulated leading to their compression.

It leads to vascular symptoms or nerve compression symptoms or both. The subclavian artery gets constricted at the site of angulation by cervical rib. Then there is an area of post-stenotic dilation where thrombus formation occurs. Parts of this thrombus break to give distal embolization (Fig. 18.17).

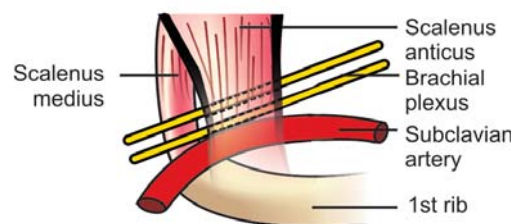


Fig. 18.16: Surgical anatomy at root of the neck

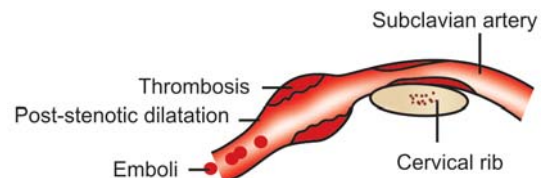


Fig. 18.17: Thrombosis and embolization from post-stenotic dilatation of subclavian artery

Clinical Features

1. *Local symptoms:* The cervical rib may be felt as a bony hard and fixed lump in the lower neck in some cases.
2. *Vascular symptoms:*
 - *Ischemic muscle pain:* Pain is felt in upper limb after movement or exercise (similar to intermittent claudication in leg).
 - Attack of pallor, coldness and cyanosis due to distal embolization. Its differentiation from Raynaud's phenomenon is difficult.
 - *Trophic changes:* Finger numbness, skin atrophy, brittle nails, muscle atrophy, ulceration, gangrene.
 - Radial pulse may be feeble or absent.
 - Systolic bruit over subclavian artery.
 - *Adson's deep breathing test:* It is based on the fact that scalenus anterior is an accessory muscle of respiration. On asking the patient to take a deep breath and turn head towards side of symptoms, radial pulse disappears due to compression of subclavian artery.
3. *Nerve pressure symptoms:*
 - Cervical rib rarely causes nerve pressure symptoms.
 - Due to compression of lower nerve roots (C_8 , T_1), ulnar nerve paresis occurs.
 - It manifests as numbness and paresthesia in the distribution of C_8 and T_1 , weakness of interosseous muscles, wasting of hypothenar muscles.

Investigations

- X-ray cervical spine to look for cervical rib.
- Arteriography for arterial compression.
- EMG and nerve conduction studies for nerve compression.

Differential Diagnosis

- *Carpal tunnel syndrome:* There is wasting of the thenar eminence due to median nerve compression (Myxoedema, Colles fracture, Rheumatoid arthritis).
- Cervical spondylitis.
- Lateral protrusion of cervical disc.
- Conditions leading to Raynaud's phenomenon.

Treatment

- Occlusion of subclavian artery without ischemia: Exercise programs to improve collateral circulation.

- Aneurysm of subclavian artery with thromboembolism and severe symptoms:
 - ☐ Excision of cervical rib with periosteum (to prevent regeneration of rib).
 - ☐ If cervical rib is not found, scalenus anterior muscle is divided (Scalenotomy).
 - ☐ Resection of the segment bearing aneurysm and thrombosis which is replaced by a graft.
 - ☐ Cervical sympathectomy (if vascular symptoms are predominant).
- Patients with mild nerve compression symptoms are relieved with exercises for strengthening muscles of shoulder girdle. Indications for surgery are severe symptoms and no response to conservative treatment.

DRUGS CAUSING GANGRENE

Ergot Preparations

- Patients suffering from migraine (vasomotor headache) taking ergot preparations over a long period of time may develop gangrene of fingertips.
- Patients taking such drugs should be well informed about the side effects since migraine is a chronic disease.
- In case of established gangrene, stoppage of drug and conservative amputation of digits may be required.

Intra-arterial Drugs

- The *drug addicts* using intravenous drugs may accidentally inject the drug in femoral artery in groin. It leads to intense pain and discoloration of the leg. Treatment consists of intra-arterial thrombolysis, intravenous dextran and heparin. In most of the cases, spontaneous resolution occurs. The cases with established gangrene need conservative amputation. There is high-risk of underlying HIV infection in these cases.
- *Thiopentone:* It is a drug given intravenously for general anesthesia. If it is accidentally injected into brachial artery, it causes severe burning pain with blanching of the hand due to intense vasospasm. The treatment is immediate injection of vasodilator drugs through the same needle lying in artery. The drugs are:
 - ☐ 2% papaverine sulphate (5 ml).
 - ☐ 1% procaine (5 ml).

Other measures are:

- ☐ Intra-arterial thrombolysis.
- ☐ Intravenous low molecular weight dextran.

If vasospasm is not relieved, gangrene of fingers may occur.

VENOUS GANGRENE

A massive deep vein thrombosis of lower limb, associated limb edema may cause limb ischemia because of impairment of blood supply. It leads to gangrene affecting foot and variable part of the leg.

The treatment is intravenous thrombolysis. A catheter is passed in affected vein and a fibrinolytic drug (streptokinase, tissue plasminogen activator) is infused.

Although the gangrene looks extensive, it involves only skin and subcutaneous tissues. Hence, limb can be salvaged in almost all the cases.

TRAUMATIC GANGRENE

A. Direct Causes

i. Crush Injury

It is seen in run over accidents where a vehicle passes over a limb. All the limb structures are badly crushed and are often non-salvageable. It leads to moist gangrene with superadded bacterial infection (Fig. 18.18). Early amputation should be done. It should be conservative amputation, i.e. only crushed tissue needs excision preserving as much limb as possible.



Fig. 18.18: Moist gangrene of dorsum of the hand in crush injury



Fig. 18.19: Bed sore occipital region



Fig. 18.20: Bed sore sacrum

ii. Pressure sores (Bed sores /Trophic ulcers)

- Bed sores are seen at pressure points (Figs 18.19 and 18.20) in patients who are bed ridden for a prolonged period of time, e.g.
 - ☐ Paraplegics due to spinal cord injury.
 - ☐ Unconscious patients due to head injury.
- These patients have definite predisposing factors (Box 18.14) that should be identified before bed sores develop.

CASE SUMMARY

20 years old male patient was admitted in comatose state following head injury. On 5th day of coma, he started running high grade fever. All investigations were normal and he did not respond to even higher antibiotics. On routine rounds, when the patient was

turned, a bedsore with local abscess was seen in sacral region. The abscess was drained and bed sore was dressed regularly (Fig. 18.20). The fever subsided thereafter.

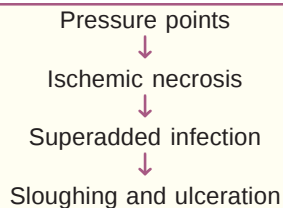
Learning point: All unconscious patients should have regular care of the back to prevent occurrence of bed sores and its complications.

Box 18.14: Bed sores: Predisposing factors

- Pressure points
- Recurrent trauma
- Moisture
- Anemia
- Malnutrition

- The mechanism of bed sore formation is shown in Box 18.15.

Box 18.15: Mechanism of bed sore formation



- To prevent bed sore, these factors should be corrected as follows:
 - ☐ Regular turning (every 3 hrs.) of the patient in the bed, to avoid pressure points.
 - ☐ To keep the bedsheet dry and wrinkle free.
 - ☐ Air beds and water beds are available for such patients that help in preventing formation of pressure points.
 - ☐ Correction of anemia by blood transfusion.
 - ☐ Correction of malnutrition by oral/parenteral nutrition.
- Once erythema develops at pressure point that does not change color on pressure, it indicates onset of bed sore.
- The area should be kept dry and covered with opposite adhesive film or with aerosol silicon spray.
- During initial stages, bed sore is treated by applying antiseptic lotion and keeping it exposed so that it remains dry.
- Once bed sore develops, treatment is regular dressing and debridement.
- Malnutrition and anemia need to be corrected.

- Once wound becomes clean, it is covered with a rotation flap.

B. Indirect Causes

It is due to arterial occlusion away from the site of gangrene (Box 18.16).

Box 18.16: Indirect traumatic gangrene: Causes

- Bone fracture compressing an artery.
- Limb injury causing arterial thrombosis.
- Limb injury causing arterial division.
- Intra-arterial injection of adrenaline containing local anesthesia.
- Compartment syndrome.

Compartment Syndrome

In closed limb injuries, there is interstitial tissue edema that leads to rise in pressure within fascial compartment. Application of tight bandages and POP caste further adds to rise in pressure. It occludes the microcirculation supplying muscles and nerves. The patient feels pain, numbness and tingling of digits. The pain is exaggerated on passive stretching of the limb muscles. The pulses are usually palpable. The condition should be diagnosed and managed early otherwise distal limb gangrene may develop.

Treatment

- In compartment syndrome:
 - ☐ Pressure dressing and castes should be removed.
 - ☐ Surgical fasciotomy is done where skin and deep fascia are incised longitudinally so that pressure on underlying structures (muscles, nerves, vessels) is released. It helps in improving circulation.
- Reduction of fracture with repair of arterial injury.
- Adrenaline containing local anesthesia should be avoided in areas lacking collateral blood supply (e.g. digits).
- Keep the limb cool to minimize the metabolism.
- Once gangrene develops, it needs conservative amputation.

PHYSICAL GANGRENE

Frostbite

- It is seen after exposure to severe cold as happens on high altitude.

- Elderly and malnourished are more prone.
- There is intense vasospasm and damage to vessel walls leading to transudation and edema.
- The patient feels severe burning pain in the limb.
- On examination, the limb appears waxy. There is formation of blisters and finally gangrene develops.

Treatment

- The limb should be gradually rewarmed.
- The patient should be kept in a warm room and given warm drinks so as to correct the generalized hypothermia.
- The limb should be covered in soft cotton wool to avoid further damage.
- Analgesics are given to relieve pain.
- Paravertebral injection in sympathetic chain may help in relieving vasospasm.
- Once gangrene develops, conservative amputation is required.

Trench Foot

- This follows exposure to extreme cold. The limb is tightly compressed with stockings, tight clothes or tight shoes.
- The condition is essentially same as frostbite.
- The tight pressure should be removed and it is treated like frostbite.

Ainhum

- It is a disease of unknown etiology.
- It is commonly seen in black males who give history of running bare foot during childhood.
- A fissure appears at the level of interphalangeal joint of 5th toe. It gradually deepens and encircles the digit.
- The digit becomes necrosed and gangrenous.
- If picked up early, it can be treated with Z-plasty.
- Once gangrene develops, treatment is amputation.

CHEMICAL GANGRENE

- Carbolic acid (Phenol) if applied to the skin leads to intense arterial spasm and gangrene.
- The affected area should be immediately washed with water and local bicarbonate soaks should be applied.
- If sloughing and gangrene occurs, it needs excision and skin grafting.

INFECTIVE GANGRENE

1. **Boil** (See Chapter 3, Infections)
2. **Carbuncle** (See Chapter 3, Infections)
3. **Cancrum Oris** (See Chapter 3, Infections)
4. **Gas Gangrene**

- It is rapidly spreading infective gangrene of the muscles caused by clostridial organisms.
- It is also called as clostridial myonecrosis.
- Clostridium is a gram positive, spore bearing organism.

The strains responsible for gas gangrene are:

- ☐ *Clostridium welchii*: Commonest (in 80% cases).
- ☐ *C. edematiens*
- ☐ *C. septicum*
- ☐ *C. histolyticum*

- Immuno-compromised patients, patients with diabetes and malignancy are at higher risk.

Pathogenesis

- Clostridial spores are present in the soil and in the patient's own fecal flora.
- In crush injuries following road side accidents, there is heavy contamination of the wound with soil.
- In surgical wounds around perineum (e.g. above knee amputation), there can be fecal contamination of the wound.
- Presence of necrotic and foreign material in the wound produces anaerobic conditions.
- The clostridia multiply in such wounds and produce several toxins (α toxin, protease, collagenase, hyaluronidase).
- The whole length of the muscle (from origin to insertion) is affected. There is necrosis of muscle with foul smelling discharge.
- There is production of gas (hydrogen sulphide, ammonia, etc.) in the wound by the organisms.
- In uncontrolled cases, septicemia can occur leading to production of gas in many organs. Formation of gas in the liver is known as **foaming liver**.
- Renal failure, circulatory failure and MOF occur if early treatment is not done.

Clinical Features

- Despite toxemia, patient is fully conscious and alert that can be misleading.
- Patient complains of intense wound pain.
- There is tachycardia and dyspnea out of proportion to fever.

- The wound appears edematous and sutures are under tension.
- On palpation, crepitus is felt due to presence of gas in subcutaneous tissue (surgical emphysema). Palpating finger gets the feeling of a 'hairy mattress'. On applying stethoscope on affected area, crackling sound is heard. Various causes of surgical emphysema are given in Box 18.17A.
- On pressing the wound margins, brownish foul smelling fluid exudes through the suture line.
- The fluid has sickly- sweet odor like that of decaying apples (mousy odor).
- X-ray shows presence of gas in subcutaneous tissue and underlying muscles.

Box 18.17A: Surgical emphysema: Causes

- | | |
|----------------------|---|
| • Traumatic | Fracture ribs
Fracture nasal air sinuses
Compressed perforating apex of tooth during dental treatment |
| • Infective | Gas gangrene |
| • Extraneous | Air entrapped during wound closure (Pseudogas gangrene) |
| • Esophageal rupture | |

Treatment

- Early and adequate treatment is required because delay can be fatal due to rapidly spreading infection.
- Fluid resuscitation and blood transfusions.
- Penicillin in high doses (2 gm 4 hrly).
- Opening of skin sutures, multiple longitudinal incisions for wide drainage.
- Aggressive surgical debridement of dead tissues.
- Limb amputation in case gangrene involves the limb.
- Hyperbaric oxygen in postoperative period helps in reducing the amount of toxin production.
- Anti gas gangrene serum (AGS) is of no proven value and is not used these days.
- Recommended measures for prevention of gas gangrene are given in Box 18.17B.

5. Fournier's Gangrene

- It is also known as idiopathic gangrene of the scrotum.
- It is a vascular disorder of infective origin.
- The hemolytic streptococcal infection is associated with staphylococcal infection.

Box 18.17B: Prevention of gas gangrene in infected wound

- Early wound debridement
 - Regular dressings
 - In compound fracture, make a window in POP cast for regular dressing
 - Prophylactic antibiotic (c penicillin)
 - Amputation of crushed and dead limb
- This synergistic infection causes severe inflammation in scrotal subcutaneous tissue leading to obliterative arteritis and gangrene formation.
 - The disease has three characteristic features:
 - i. Sudden appearance of scrotal inflammation.
 - ii. Rapid onset of gangrene.
 - iii. Absence of other usual causes of gangrene.
 - There is sudden severe pain in scrotum along with fever and malaise.
 - Scrotal edema appears and within hours or days, it changes to scrotal gangrene (Fig. 18.21).
 - *Treatment:*
 - ☐ Antibiotics
 - ☐ Scrotal incision during stage of edema.
 - ☐ Wide excision of scrotal skin once gangrene develops.

6. Meleney's Gangrene (Pyoderma Gangrenosum)

- It usually affects abdominal or chest wall after operation for a septic condition.
- There is synergistic infection caused by streptococci and staphylococci (similar to Fournier's gangrene).



Fig. 18.21: Fournier's gangrene scrotum

- It is also known as '*postoperative bacterial synergistic gangrene*'.
- An area of cellulitis appears that rapidly progresses to formation of gangrene.
- *Treatment*:
 - ☐ Antibiotics.
 - ☐ Metronidazole.
 - ☐ Hyperbaric oxygen.
 - ☐ Opening of suture line and wide drainage.
 - ☐ Wide excision once gangrene develops.

DIABETIC GANGRENE

- The diabetic foot is at a greater risk of infection even from minor injuries.
- Once infection occurs, there is rapid dramatic deterioration leading to gangrene formation that requires amputation of the foot.
- The incidence of gangrene in diabetics is fifty times more than in nondiabetics.

Pathophysiology

Following factors are responsible for gangrene formation in the diabetic foot:

Peripheral Neuropathy

- Diabetic patients have typically symmetrical sensory neuropathy affecting all the four limbs (glove and stocking type). There is loss of light touch, pain and vibration sensations and absent ankle reflexes.
- Due to motor neuropathy, there is wasting of small muscles of foot causing deformities (claw or hammer toes) and formation of pressure sores.
- Due to sympathetic neuropathy, there is dryness and vasodilatation of skin making it more prone to infections.

Thus, neuropathic foot is more susceptible to heat, chemical and mechanical trauma.

Peripheral Vascular Disease

There is atherosclerosis of arteries leading to limb ischemia.

Poor Wound Healing

Hyperglycemia provides enriched growth media for bacteria. Also there is reduced phagocytic activity of leukocytes that affects wound healing.

Box 18.18: Diabetic gangrene: Causes of death

- Uncontrolled sepsis.
- Multiorgan failure.
- Diabetic ketoacidosis.
- Electrolyte imbalance.
- Chronic debility and malnutrition.

Course of Events in Diabetic Foot

- Foot ulcers usually start at pressure areas such as first and fifth metatarsophalangeal joints, heel and pressure points due to ill fitting shoes.
- Ulcers get infected leading to cellulitis (Fig. 18.22).
- Rapid spread of infection along subfascial planes and tendon sheaths.
- Infection of bones leading to osteomyelitis.
- Severe sepsis alongwith arterial insufficiency produces wet gangrene affecting toes and foot.
- Uncontrolled sepsis leads to septicemia and death (Box 18.18).

CASE SUMMARY

60 year old male presented with history of diabetes for 10 yrs, controlled with oral hypoglycemics. One week ago, he sustained minor trauma on left big toe and developed a small wound. His blood sugar shot up to 300 mg% and he developed painless swelling and redness of big toe that started spreading rapidly. He took antibiotics from a local practitioner and continued with oral hypoglycemics. The swelling and



Fig. 18.22: Diabetic gangrene of big toe with spreading cellulitis

redness extended to involve foot and lower leg with foul smelling discharge from big toe (Fig. 18.22). He presented in emergency with high fever and drowsiness. His pulse was feeble and blood pressure was low. He was admitted with diagnosis of diabetic gangrene foot and septicemia. He was put on injection insulin, parenteral broad spectrum antibiotics and resuscitated with IV fluids and blood transfusion. Fasciotomy of left foot and leg was done under spinal anesthesia. However he didn't respond and cellulitis kept spreading up. He underwent below knee amputation as a life saving measure. However his condition kept on deteriorating and he developed anuria and jaundice suggestive of multiorgan failure. He died on 10th day of his hospitalization.

Learning point: This case emphasizes that even a minor trauma can be fatal in a diabetic patient and should not be taken lightly.

Examination

General Examination

Look for systemic manifestations of diabetes mellitus (Box 18.19).

Box 18.19: Systemic features of diabetes mellitus

- Retinopathy—blindness
- Nephropathy—renal failure
- Neuropathy—diabetic foot ulcer/ gangrene
- Cardiovascular disease—angina, Infarction
- Cerebrovascular disease—hemiplegia (CVA)
- Peripheral vascular disease—chronic limb ischemia

Local Examination

- Look for local swelling/ulcer, redness and extent of wet gangrene.
- Pain on deep palpation indicates underlying pus.
- Palpation of peripheral pulses (dorsalis pedis, posterior tibial) to look for arterial occlusion.
- In case of septicemia, patient may have fever, tachycardia, hypotension and altered sensorium.

Investigations

- Urine examination for sugar and ketone bodies.
- Blood sugar (fasting and post prandial).
- Estimation of glycosylated hemoglobin is a sensitive test for diabetes.

- Total and differential leukocyte count. Polymorphonuclear leukocytosis suggests presence of sepsis.
- X-ray of foot and leg to look for any osteomyelitis.
- Pus swab for culture and sensitivity.
- If peripheral pulses can not be palpated due to limb edema, Doppler ultrasound is used to look for the blood flow in the peripheral vessels.
- Other investigations for routine work-up (blood urea, serum creatinine, serum electrolytes, ECG, chest X-ray).

Treatment

- *Control of diabetes* by drugs (oral hypoglycemics, insulin) and diet control. In presence of sepsis, the diabetes gets worsened. Hence, patient with foot sepsis/ diabetic gangrene should be hospitalized and treated with injection crystalline insulin given subcutaneously three times a day (Box 18.20). The dosage is titrated based on urine sugar and blood sugar levels. While patient is getting insulin, it is important to watch for sudden hypoglycemia that can even be fatal sometimes. The patient should be instructed to keep sugar on his bedside and in case of giddiness, sweating, pallor and weakness (features of hypoglycemia), he should swallow a tablespoon of sugar immediately.

Box 18.20: Control of diabetes with insulin—sliding scale method

Color of uristix	Dose of plain insulin
• Blue	Nil
• Green	4 units
• Yellow	8 units
• Orange	12 units
• Red	16 units
• Red precipitates	20 units

- *Control of sepsis* with broad spectrum intravenous antibiotics (cephalosporin, aminoglycoside and metronidazole). The drugs can be later modified based on culture and sensitivity reports.
- *Early surgical drainage* and debridement of all dead tissues (Box 18.21).
- Rapidly spreading infection requires wide drainage by longitudinal incisions involving skin and deep fascia of the leg (fasciotomy).

Box 18.21: Diabetic foot—surgical treatment

• Abscess	Incision and drainage
• Sloughed ulcer	Debridement and regular dressing
• Spreading cellulitis	Multiple fasciotomy
• Gangrene	Amputation

- Once gangrene occurs, it requires *amputation*. The type of amputation depends upon extent of gangrene:
 - i. *Digital amputation*: Amputation of an isolated gangrenous digit.
 - ii. *Ray amputation*: When digital gangrene extends to involve adjoining metatarsal head, it requires excision of digit along with metatarsal head. The wound should be left open and dressed regularly to control sepsis.
 - iii. *Transmetatarsal amputation*: When infection involves more than one digit and extends on to the dorsum of foot.
 - iv. *Below knee/Above knee amputation*: If infection

and gangrene spreads to involve leg/ thigh and there is risk of septicemia.

- General measures like intravenous fluids to correct dehydration, high protein diet to improve nutrition, blood transfusion to correct anemia and physiotherapy to prevent chest infection and bed sore formation.
- *Prevention of diabetic foot gangrene*:
 - i. Patient education
 - ☐ Proper hygiene of the feet.
 - ☐ Regular inspection of the feet.
 - ☐ Use well fitting footwear and never walk bare-feet.
 - ☐ Use nail cutter (not blade) for nail trimming.
 - ☐ Consult surgeon even in case of minor foot trauma.
 - ii. Good diabetes control by a physician.
 - iii. Correction of bony deformities (exostosis, hammer toe) by orthopedic surgeon.

Thus, multidisciplinary approach is needed to take care of diabetic foot patients.

19

Chapter

Diseases of Venous System

Sanjay Marwah

- Venous diseases mainly affect lower limb veins.
- The important diseases are:
 - ☐ Varicose veins
 - ☐ Deep vein thrombosis

Surgical Anatomy of the Lower Limb Venous System

- Venous return from the lower limb is through superficial and deep veins.
- The superficial veins are superficial to deep fascia while the deep veins are deep-to-deep fascia.
- Most of the blood in lower limbs (90%) is carried in deep veins.
- The principal superficial veins are long and short saphenous veins and are the site of varicose veins (Box 19.1).
- The principal deep veins are anterior tibial, posterior tibial and peroneal veins in calf (soleus plexus of veins), popliteal vein in knee and femoral vein in thigh region. These are the sites of deep vein thrombosis.
- The short saphenous vein starts posterior to lateral malleolus and ends at saphenopopliteal junction in popliteal fossa draining into deep venous system (Fig. 19.1).

Box 19.1: Long saphenous vein vs short saphenous vein

	Long saphenous vein	Short saphenous vein
Origin	Front of medial malleolus	Behind lateral malleolus
Ends at	Groin (sapheno-femoral junction)	Popliteal fossa (Saphenopopliteal junction)
Related nerve	Saphenous nerve	Sural nerve

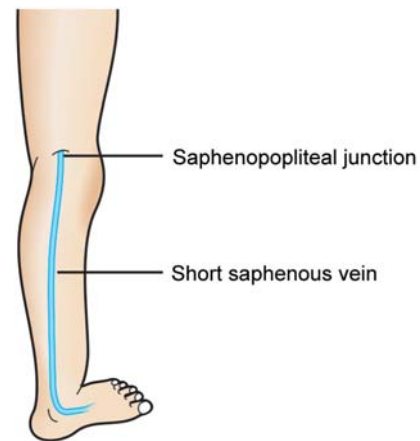


Fig. 19.1: Course of short saphenous system on back of the leg

- The long saphenous vein starts anterior to medial malleolus, ascends on postero-medial side of knee and ends at saphenofemoral junction (SF) in upper thigh draining in to deep venous system. This junction lies 4 cm below and lateral to pubic tubercle.
- In the leg, saphenous nerve accompanies the long saphenous vein and sural nerve accompanies the short saphenous vein.
- Superficial venous system also drains into deep veins at ankle, calf and thigh through perforating veins. These perforators have valves which prevent back flow of blood from deep to superficial veins.
- There are three perforators in lower leg (**Cockett**), one just below knee (**Boyd**) and one in mid thigh (**Dodd**) (Fig. 19.2).
- All superficial and deep veins have valves to prevent back flow of blood.

Surgical Physiology

The blood normally flows from superficial veins to deep venous system. On walking and exercise, calf and thigh

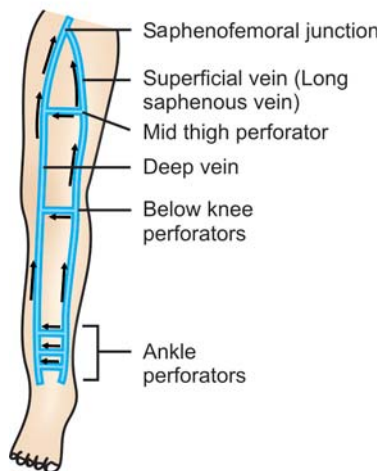


Fig. 19.2: Superficial, deep and perforating veins of the lower limb. Normally blood flows from superficial to deep veins via perforators

muscles contract compressing the deep veins and pumping blood towards the heart. Valves prevent back flow of blood from deep veins to superficial veins. If valves become incompetent, the muscle contraction will cause reverse flow of blood through perforating veins into superficial veins giving rise to varicose veins.

VARICOSE VEINS

A varicose vein may be defined as a vein that becomes elongated, dilated, tortuous and thickened due to continuous dilatation under pressure (Fig. 19.3). Risk factors for development of varicose veins in lower limbs are given in Box 19.2.

Various sites where varicosities can develop in the body are given in Box 19.3.



Fig. 19.3: Varicose veins right lower limb

Box 19.2: Risk factors for varicose veins

- Familial
- Female gender
- Advancing age
- Prolonged standing
- Obesity

Box 19.3: Varicosities—sites

- Long saphenous vein
- Short Saphenous vein
- Varicocele
- Hemorrhoids
- Esophageal varices

Primary Varicose Veins

These are varicose veins which develop due to intrinsic valvular and saphenofemoral incompetence and have no apparent underlying cause.

Secondary Varicose Veins

These are varicose veins which develop due to some underlying pathology.

- Obstruction to venous outflow by:
 - ☐ Gravid uterus (pregnancy)
 - ☐ Pelvic tumors (uterus, ovary, cervix, rectum)
 - ☐ Iliofemoral thrombosis
 - ☐ Inguinal/iliac lymphadenopathy
- Valve destruction (due to deep vein thrombosis).
- Arteriovenous fistula (traumatic or congenital).

Clinical Features

Symptoms: There is dull ache in the calf and lower leg due to pooling of blood in superficial veins. These symptoms worsen by evening and often accompanied by ankle swelling. Elevation of feet relieves the symptoms.

Patient may be asymptomatic and complain about the cosmetic appearance only. Patient may present with some complication of varicose vein, e.g. eczema, pigmentation, ulceration, etc.

A clinical classification of varicose veins is given in Box 19.4.

Clinical Examination

- Inspection of the affected lower limb should always be done in *standing position*. Examine the limb from

Box 19.4: Varicose veins: Clinical classification

Grade	Features
0	No visible or palpable sign of venous disease.
1	Telangiectasia, reticular veins or malleolar flare.
2	Varicose veins.
3	Cutaneous edema without skin changes.*
4	Skin changes.*
5	Skin changes* with healed ulcer.
6	Skin changes* with active ulcer.

*Skin changes: Eczema, pigmentation or lipodermatosclerosis.

umbilicus to toe looking front as well as back of the limb. Look for the dilated veins involving long or short saphenous system. Also look for complications of varicose veins especially in lower leg in form of edema, pigmentation, dermatitis, ulceration.

- Palpation of lower abdomen and pelvis to look for any cause of secondary varicose veins.
- Clinical tests are done with following three aims:
 - ☐ To know which venous system is involved (long or short saphenous system).
 - ☐ To look for perforator incompetence.
 - ☐ To look for patency of deep veins.
- If long saphenous system is involved, varicose veins are seen anterior to medial malleolus, on medial side of leg and thigh up to SF junction.
- If short saphenous system is involved, varicose veins are seen posterior to lateral malleolus, on back of the leg up to saphenopopliteal junction in popliteal fossa.
- *Schwartz test*: With the patient in standing position, two fingers of left hand are placed on SF junction and right index finger taps the most prominent part of varicosity below in the leg. A palpable fluid thrill felt by the fingers at SF junction suggests superficial column of blood in the veins.
- *Cough impulse test*: In standing position, examining finger is kept at SF junction and the patient is asked to cough. A palpable fluid thrill suggests sapheno-femoral incompetence.
- *Trendelenburg test*: It is done in two parts, one is to detect perforator incompetence and second is to detect SF incompetence. The patient is asked to lie supine on bed and lower limb is elevated to empty the dilated veins. SF junction is occluded with thumb

and patient is asked to stand quickly. After this, following two steps are followed:

Trendelenburg I: The thumb pressure is immediately released on standing. If there is quick filling of veins from above downwards, it is suggestive of SF incompetence.

Trendelenburg II: The pressure with thumb is maintained at SF junction. If there is gradual filling of veins from below upwards it is suggestive of incompetent perforators that allow retrograde flow of blood.

For short saphenous system, a similar test can be performed with thumb pressure at saphenopopliteal junction.

- *Multiple tourniquet test*: Patient is asked to lie supine on bed and limb is elevated to empty the veins. Four tourniquets are applied to occlude the SF junction and perforators at three levels (mid thigh, below knee and above ankle). The patient is asked to stand up and tourniquets are released one by one from below upwards. Sudden filling of veins on release of a tourniquet indicates the level of incompetent perforator.
- *Fegan's method*: It is used for locating the site of incompetent perforators. In standing position, dilated veins are marked with a marking pen. The patient is asked to lie down and the affected limb is raised to empty the vein. Palpate the line of marked varicosity carefully. The incompetent perforators are felt as circular openings with sharp edges due to gaps created in deep fascia.
- *Modified Perthes' test*.* It is done to test the patency of deep veins. In standing position, a tourniquet is tied at SF junction and the patient is asked to walk briskly for five minutes. If patient complains of bursting pain in the leg and superficial varicosities become more prominent, it indicates that deep veins are occluded. Positive Perthes' test is absolute contraindication for surgery of varicose veins.

Investigations

In case of smaller varices, the clinical tests may give equivocal results requiring investigations as follows:

- *Hand held Doppler ultrasound probe* is very useful in detecting the level of incompetence. Today, it is

*In original Perthes' test, the patient was asked to walk after wrapping the limb with elastic bandage.

Box 19.5: Indications for intervention

- Cosmetic appearance
- Medical fitness for a job (police, army, etc.)
- Severe leg cramps
- Complications (ulcer, eczema, bleeding, etc.)

the minimum investigation required for treating patients with varicose veins.

- *Duplex ultrasound imaging* gives direct visualization of veins. It gives anatomical as well as functional information.
- *Venography*: It is an invasive investigation. With availability of duplex scanning, it is usually not required these days.

Treatment

- The indications for intervention in a case of varicose veins are given in Box 19.5.
- The modalities of treatment for varicose veins are:
 1. Conservative
 2. Sclerotherapy
 3. Surgery
- 1. *Conservative treatment*: Indications for conservative treatment are:
 - Old age
 - Patient unfit for surgery
 - Secondary varicose veins (pregnancy, pelvic tumors)
 - Deep vein thrombosis

Measures taken in conservative treatment are:

- Avoid prolonged standing.
 - Elastic stocking during the day.
 - Elevation of the leg during night.
 - Exercise of leg muscles.
 - Drugs—calcium dobesilate.
2. *Sclerotherapy*: Injection sclerotherapy is used for venous blowouts and small below knee varicosities (up to 2 mm) without perforator incompetence. It promotes inflammatory reaction followed by obliteration of varicosity. A fine needle is used to inject sclerosant (sodium tetradecyl sulphate) into the lumen of varicosity. A compression stocking is applied to maintain pressure for one week. Indications of sclerotherapy are given in Box 19.6. Complications of sclerotherapy are given in Box 19.7.

Box 19.6: Indications of sclerotherapy

- Hemangioma
- Varicose veins (small)
- Hemorrhoids
- Esophageal varices

Box 19.7: Complications of sclerotherapy

- Anaphylactic reaction
- Skin pigmentation
- Skin ulceration (if drug injected in subcutaneous tissue)
- Deep vein thrombosis (if drug spreads to deep veins)
- Thrombophlebitis

3. Surgery:

- Surgical treatment is used to remove large varicosities of main venous trunks (long and short saphenous veins) and small varicosities (2-3 mm size).
- Preoperative localization of venous anatomy and perforators should always be done with Doppler ultrasound and marked with skin ink.
- For saphenofemoral junction incompetence, saphenofemoral ligation (**Trendelenburg procedure**) is done.
- For saphenopopliteal junction incompetence, saphenopopliteal ligation is done.
- However, simple ligation of these sites is associated with high rate of recurrence due to reflux through communications between superficial and deep venous system.
- Stripping of long or short saphenous vein significantly reduces the recurrence rate by disrupting the perforators connecting superficial and deep venous system.
- However, removal of saphenous veins by stripping has risk of injuring the nerves accompanying them.
- To avoid injury to saphenous nerve at ankle, the long saphenous vein should be removed up to mid calf level and not up to medial malleolus (as was the practice earlier).
- To avoid injury to sural nerve, great care should be taken in removing short saphenous vein in leg.
- Incompetent perforators are explored, identified deep to deep fascia and ligated subfascially (**Cockett and Dodd operation**).
- Small varicosities are explored in subcutaneous tissue, ligated and removed.

Box 19.8: Complications of varicose veins surgery

- Hematoma
- Wound infection
- Nerve injury (long saphenous and sural nerve)
- Major vascular injury (femoral and popliteal artery)

- Complications of varicose veins surgery are given in Box 19.8.

Operative Techniques*Trendelenburg Procedure*

An inguinal incision is made to expose SF junction. Three tributaries draining into long saphenous vein are identified, ligated and divided to prevent recurrence through collateral drainage. Long saphenous vein is ligated flush with femoral vein.

Stripping of Long Saphenous Vein

After ligating and dividing the long saphenous vein in groin (as described above), an olive tip Myer's vein stripper is passed down the long saphenous vein and its end is identified through skin in upper calf. A small skin incision is made to expose the stripper. The stripper is pulled through calf incision and long saphenous vein is avulsed. Skin incisions are sutured and tight crepe bandage is applied.

Stripping of Short Saphenous Vein

Unlike SF junction, saphenopopliteal junction is variable. Hence, it should be localized with Doppler ultrasound preoperatively. The junction is exposed and divided. A stripper is passed down the short saphenous vein and recovered at mid calf level. The vein is avulsed and removed. To avoid injury to sural nerve, a pin-stripper is used instead of olive tip stripper for removing short saphenous vein.

New Surgical Techniques

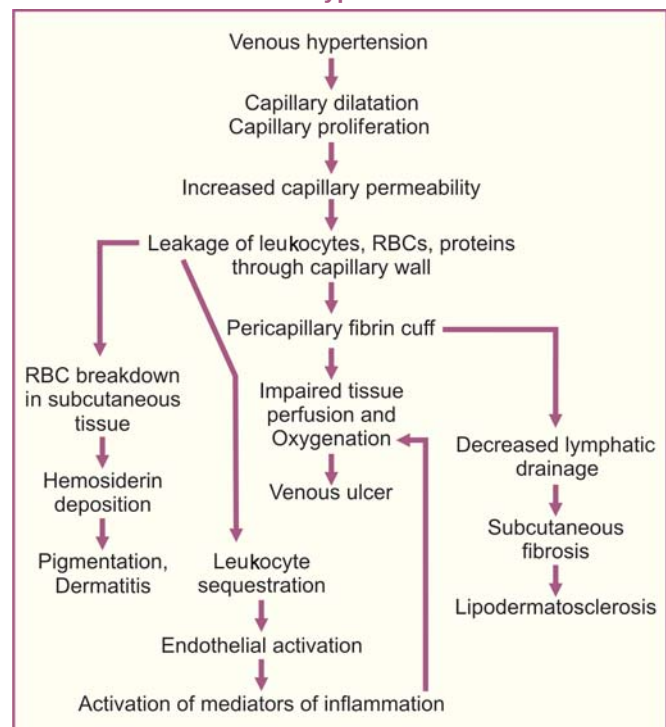
- *Radiofrequency ablation or Laser ablation of the saphenous vein:* Instead of stripping, intraluminal destruction of saphenous vein using ablation catheter helps to minimize postoperative discomfort. The catheter is inserted into the vein up to SF junction under ultrasound guidance. Catheter is gradually

withdrawn and alternating current is applied for rapid thermal electro-coagulation of the vein.

- *Subfascial Endoscopic Perforator Surgery (SEPS):* Endoscopic instruments are placed through small skin ports at distant sites for interruption of incompetent perforators. Thus, it is possible to ligate incompetent calf perforators without need of making incisions in region of scarred skin of lower leg that heals very poorly.
- *Hook phlebectomy:* It is used for removal of small varicosities. Instead of long skin incision, 1-2 mm incision is made and small hooks are used to deliver the varicosity to surface where it is ligated and excised. The incision does not require any suture and cosmetic outcome is excellent.

Complications of Varicose Veins

In long standing varicose veins, refluxing blood from the deep to superficial venous system during muscle contraction leads to venous hypertension. The venous hypertension causes damage to skin and subcutaneous tissue (Box 19.9).

Box 19.9: Pathophysiology of damage caused by venous hypertension

Various complications are:

Pigmentation

Dark brown discoloration is seen in lower third of leg and around ankle. It is due to RBC breakdown and hemosiderin deposition in subcutaneous tissue.

Dermatitis

Areas of redness and itching appear in lower leg due to hemosiderin deposition. Local application of ointments may also add to allergic skin manifestations.

Lipodermatosclerosis

Various skin changes in form of eczema, pigmentation, subcutaneous fibrosis etc. seen in lower leg are called as lipodermatosclerosis (Fig. 19.4). Its mechanism is shown in Box 19.9.

Thrombophlebitis

It is inflammation of superficial veins. The veins become red, tender and feel as cord like swelling in subcutaneous tissue.

Ulceration

The ulcer is situated in lower leg above medial malleolus and is associated with varicose veins and lipoder-



Fig. 19.4: Bilateral varicose veins with lipodermatosclerosis

matosclerosis. It is called as '*varicose ulcer*' or '*gravitational ulcer*'. The ulcer is irregular shaped with shelving edges, shallow depth (never penetrates deep fascia) and floor is covered by slough or granulation tissue (see Fig. 5.11). Most of the times, ulcer is painless but severe infection or involvement of saphenous nerve can cause pain in the ulcer. Sometimes ulcer may become large and involve circumference of the leg.

Rarely venous ulcer may occur following deep vein thrombosis and the patient presents with bursting pain in the limb along with leg swelling. The swelling involves upper leg and there is extensive scarring and ulceration in lower leg. This is characteristically described as '*inverted beer bottle*' appearance.

The pathophysiology of ulcer formation is shown in Box 19.9.

In varicose ulcer, Doppler ultrasonography should be done to delineate superficial veins, perforators and deep veins.

Treatment of Varicose Ulcer

Conservative treatment: It is called as *Bisgaard regime*. It includes:

- Limb elevation at night.
- Active and passive leg exercises to contract calf muscles.
- Correct way of walking with heel down first.
- *Compression stocking* covering ankle and the leg. Most patients of venous ulcer respond to compression treatment. Compression treatment should be continued even after ulcer heals since it helps in preventing ulcer recurrence.
- In case of *infected ulcer*, treatment is local cleaning, debridement and regular dressing. Topical antibiotics are ineffective and cause skin allergy. In case of local dermatitis, topical steroids should be applied. Systemic antibiotics may be required in case of cellulitis, lymphangitis and systemic sepsis.
- *Drugs* used for venous ulcer are pentoxifylline, diosmin and prostaglandins. But none of these have been found to be effective.

Surgical Treatment

- Varicose veins need stripping.
- Incompetent perforators need subfascial ligation.
- Endoscopic surgery (SEFS) is ideal in such cases since it avoids incision in scarred skin of lower leg.

- In deep venous insufficiency, reconstruction of deep valves (valvuloplasty) has no definite role and treatment is conservative.

Marjolin's Ulcer

In a few neglected cases of venous ulcer, squamous cell carcinoma may develop. The margins become everted and it starts growing in size (see Fig. 5.18). It is essential to take biopsy from any ulcer with everted margins. The groin lymph nodes should always be examined to look for enlargement due to metastasis.

Hemorrhage

Minor trauma over a dilated vein can cause torrential bleeding. It is controlled by limb elevation and pressure bandage. A tourniquet should never be used to control venous bleeding.

Calcification

In long standing cases, the walls of dilated veins may get calcified.

Periostitis

If ulcer is situated over medial side of leg on tibia, it involves periosteum and causes severe pain.

Foot Deformity

In long standing cases of periostitis, talipes equinus deformity occurs because patient walks on the toes to get relief from pain. It leads to contracture and shortening of tendo-Achillis.

DEEP VEIN THROMBOSIS

- There is formation of blood clot in deep veins and it commonly affects the legs (soleus plexus).
- It can cause sudden death due to pulmonary embolism.
- Locally, it can cause chronic venous insufficiency and venous ulceration.
- *Etiology* of DVT is described by *Virchow's triad*:
 1. Damage to vessel wall
 2. Decreased blood flow
 3. Increased coagulability.
 Increased coagulability of blood is most important factor of the triad. Its causes can be:

Box 19.10: Deep vein thrombosis—causes

T	Trauma—iatrogenic injury damaging vessel wall
H	Hormones—oral contraceptives
R	Road side accident
O	Operation—splenectomy, pelvic surgery
M	Malignancy
B	Blood dyscrasia—sickle cell anemia
O	Obesity
S	Serious co-morbid illness—diabetes, hypertension
I	Inherited—protein C and protein S deficiency
S	Sepsis

Learn causes from the word '**THROMBOSIS**'

- Inherited*: Antithrombin deficiency, protein C deficiency, protein S deficiency and presence of factor V Leiden.
 - Acquired*: Malignancy, immobilization, major surgery, sepsis, oral contraceptives, obesity, polycythemia.
- Various causes of DVT are summarized in Box 19.10.

Clinical Features

- The leg involved in DVT may be asymptomatic.
- In symptomatic case, there is pain and swelling of the leg (Fig. 19.5).
- Low grade fever.
- Tenderness in the calf muscles.



Fig. 19.5: Deep vein thrombosis left leg

- Signs:
 - 'Homan's sign': Dorsiflexion of the ankle causes pain in the calf.
 - 'Moses sign': Squeezing of calf muscles is painful. However, these clinical signs should not be elicited since they can cause dislodgement of thrombus leading to pulmonary embolism.
- Phlegmasia Alba Dolens* (Painful white leg): It is seen when thrombus extends up to iliofemoral vein.
- Phlegmasia Cerulæ Dolens* (Painful blue leg): It is seen in massive thrombosis of iliofemoral vein. There is severe pain in limb and it becomes greatly swollen and cyanotic. It may result in rise in hydrostatic pressure that causes arterial compression and venous gangrene.
- Formation of leg ulcer (Post-thrombotic ulcer).

Investigations

- Doppler ultrasound*: It detects presence of thrombus in deep veins. Being noninvasive, it is investigation of choice.
- Ascending venography*: It is not used since it is invasive.
- Magnetic resonance venography (MRV)*: It is noninvasive and differentiates between fresh and old thrombus.
- Contrast enhanced CT scan of lungs* is investigation of choice for detecting pulmonary embolism.
- D-dimer estimation*: It is degradation product of fibrin molecules and its level rises in DVT due to breaking of blood clot. If negative, it indicates absence of DVT. However, positive values can be seen in various other conditions (e.g. DIC, malignancy, infections, etc.) apart from DVT.

Differential diagnosis of DVT is shown in Box 19.11.

Treatment

- Limb elevation to reduce swelling.
- Anti-inflammatory and analgesic drugs.
- Anticoagulants*: It should be started early even on clinical suspicion of DVT so as to prevent pulmonary embolism. Heparin is given intravenously. Initial adult dose is 10000 units followed by 5000 units 8 hourly given for 5 days. During heparin therapy, its dose is regulated by activated partial thromboplastin time (APTT) value that is kept 2-2½ times

Box 19.11: Differential diagnosis of DVT

- Cellulitis
- Trauma (Hematoma of calf muscle)
- Lymphatic obstruction
- Myxoedema
- Congestive heart failure
- Nephrotic syndrome
- Superficial thrombophlebitis

of normal value. At the same time, oral anticoagulants (warfarin) are started and continued for 3-6 months. Warfarin dosage is controlled by measuring international normalized ratio (INR) that should be prolonged to 2-2½ times of control value.

An alternative and better method of anticoagulation is to give low molecular weight heparin (LMWH) by subcutaneous injection. The advantage is that dosage is once daily and blood test monitoring is not required. With LMWH, warfarin treatment is started in the same way as with heparin.

Complications and prevention of DVT are shown in Boxes 19.12 and 19.13 respectively.

Box 19.12: Complications of DVT

- Pulmonary embolism
- Chronic venous insufficiency
- Venous ulcer
- Varicose veins
- Venous gangrene

Box 19.13: Prevention of DVT in patients undergoing surgery

Preoperative	Weight reduction in obese patients Stop oral contraceptives Adequate hydration
Intraoperative	Proper patient positioning to avoid pressure on calf Intermittent pneumatic compression of calf Compression stockings LMWH therapy Dextran 70 to prevent red cell aggregation
Postoperative	Early mobilization Chest physiotherapy Adequate analgesia Hydration

20 Chapter

Principles of Operative Surgery, Diathermy, Radiotherapy and Anesthesia

Sanjay Marwah, Naveen Malhotra

PRINCIPLES OF OPERATIVE SURGERY

Every surgeon has his own particular way of doing operations. However, there are some basic rules of operative techniques that should be learnt by the beginners.

Skin Incision

- The skin incision should be planned in a way that it gives good view of the structures to be operated.
- The skin incision should be given in natural skin crease (Lines of Langer) so that final scar is less visible and gives good cosmetic result (see Fig. 6.9).
- The skin incision should avoid damage to important underlying structures like nerves and vessels. So incision should be parallel and not across the long axis of these structures.
- The skin incision is made using a scalpel with a blade. The blade has a curved margin and it has stabbing and shearing actions. A little of both the actions is required in most surgical procedures.
- The blades are available in various shapes and sizes (Fig. 20.1) and are chosen depending upon a particular procedure, e.g.
Number 11: For making skin hole to put a drain or for arteriotomy.
Number 15: For curved incisions and fine dissection.
Number 10, 22, 23: For long, straight incisions.
- For holding scalpel, two grips are used:
 - **Pen grip** that permits fine angulations (Fig. 20.2A).
 - **Stroke grip** that permits knife to be used with some force (Fig. 20.2B).
- The skin should be cut cleanly in one stroke with plane of the blade held perpendicular to skin surface. At the same time, tension should be applied on the

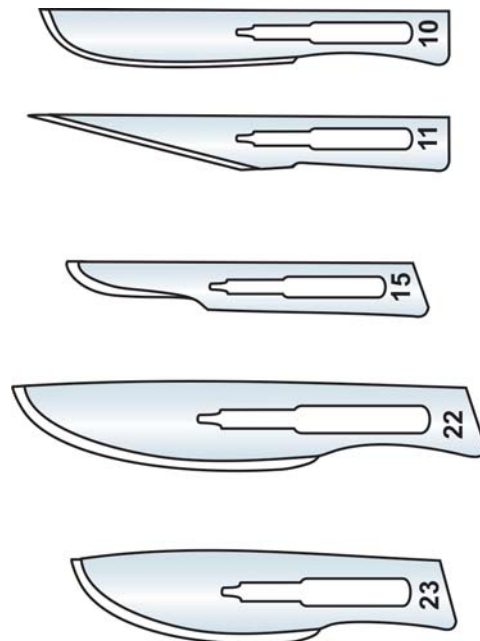


Fig. 20.1: Scalpel blades—shapes and sizes

skin across the line of incision so that the skin divides cleanly (Fig. 20.3).

Dissection

- The basis of soft tissue dissection is that tissues are placed under tension during dissection (one cannot dissect a jelly). It is achieved by holding and lifting the tissues with dissecting forceps.
- The tissue dissection can be **sharp** or **blunt**. **Sharp dissection** is done with scissors, knife or diathermy. These days diathermy dissection is used more often as it reduces blood loss, saves operating time and also appears to reduce postoperative pain.



Fig. 20.2A: 'Pen grip' for holding the scalpel

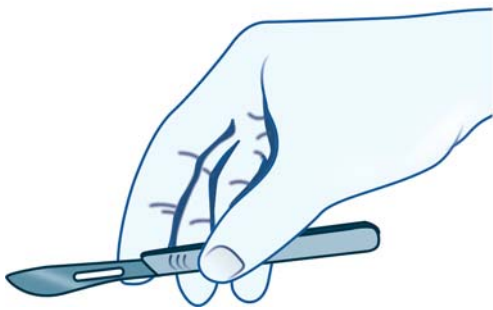


Fig. 20.2B: 'Stroke grip' for holding the scalpel

Blunt dissection is done with a cotton pledget held with Kocher's forceps or artery forceps.

- Blunt dissection is usually safer but sharp dissection is more precise and less traumatic.

Hemostasis

- Small superficial vessels are generally occluded by firm and uniform pressure alone.
- Bleeding vessels (≥ 1 mm) need to be secured with artery forceps and coagulated with diathermy current.
- Larger vessels that cross the line of incision should be identified and clamped between two artery forceps before they are divided. After dividing the vessel, it is ligated with a fine suture (Fig. 20.4). In critical vessels, it is wise to apply double ligature.
- As an alternative to ligature, fine titanium clips (hemoclips) may be applied to secure vessels using a clip applicator. These are useful for vessels lying in depth or during laparoscopic surgery.
- Sometimes there is diffuse venous bleeding and the patient's condition is too serious to allow further



Fig. 20.3: Tension applied on skin during incision

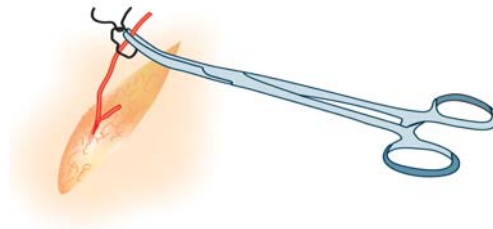


Fig. 20.4: Suture ligation of a vessel clamped in an artery forceps

dissection and additional blood loss. In such a situation, tight packing is done with roll gauze. The pack is usually removed after 48 hours and bleeding stops by that time.

- Local hemostatic agents like 'Surgicel', 'Gel foam' and 'Fibrin glue' can be used to control generalized oozing points. 'Bone wax' is useful in controlling oozing from bone edges.

Wound Closure

- The wound should be closed after achieving hemostasis and obliterating dead spaces.
- If there is dead space and possibility of oozing in postoperative period, wound should be drained with a tube drain taken out through a separate stab incision. It is connected to a drainage bag making it a closed drainage system (Fig. 20.5).
- For skin sutures, needle is inserted at right angle to skin surface so as to avoid inversion of the edges.



Fig. 20.5: Closed suction drain (after thyroidectomy) connected to a drainage bag

- The sutures are tied with only sufficient tightness to bring skin edges together without constriction. If wound is closed too tightly, it will cause tissue ischemia and wound edge necrosis.
- As suture is tightened, the knot should be drawn to one side of the wound. The final throw of knot should be tightened to prevent slipping.
- The end of the suture should be cut long enough to give an easy grasp at the time of suture removal.
- The gap between skin stitches should be twice the thickness of the skin.
- In case of zig zag wound, sutures should be put at the tip of the corner first so as to avoid 'dog-ear' formation.
- Methods of skin suturing—See chapter 28: Surgical Suturing.
- The skin sutures should be removed as soon as wound is healed (Box 20.1). Delay in suture removal causes more scarring and infection due to presence of foreign body.

Box 20.1: Time of suture removal

• Face and neck	3 days
• Scalp	5 days
• Upper limb and groin	7 days
• Abdomen	10 days
• Lower limbs	14 days

- The suture is cut at a site where it enters the skin (Fig. 20.6). Thus, on pulling, exposed part of

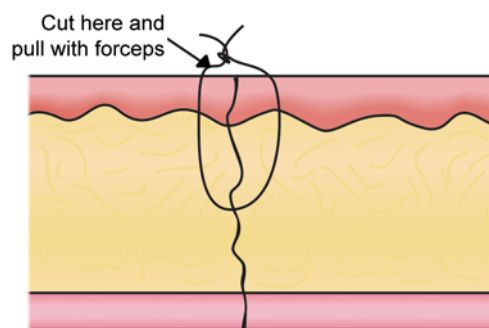


Fig. 20.6: Method of cutting and suture removal

suture does not traverse the suture tract and there is no risk of skin organisms entering the suture tract.

- Skin staplers can be used to apply staples for skin closure. It shortens the operating time. Also the skin heals without crossmarks since staples produce minimal tissue reaction. However, chances of hematoma formation are more since it is less hemostatic.
- Cyanoacrylate is surgical adhesive that can be used for skin closure.

PRINCIPLES OF DIATHERMY

It is an electrosurgical device that has become the most important and basic surgical tool in the operating room. Its application is based upon the fact that electricity passing through a conductor heats it. When the pathway for the current is very wide, the heating effect is negligible. When it is made small, one may obtain any desired amount of heat that produces mild coagulation to tissue cutting, simply by increasing the current.

If direct current is used, the patient will jump each time the current is switched on and off.

If alternating current (such as obtained from AC mains) is used, it will produce repetitive contractions giving pain and sustained tetany.

If the frequency of alternating current is increased above 10 kHz (10000 cycles/sec), then there is neither pain nor muscle stimulation. In such situation, it becomes possible to make use of heating effect of narrow electrode for either coagulation or cutting tissues.

Thus, high frequency alternating current can be delivered in either a *monopolar* or *bipolar* fashion. The



Fig. 20.7: A diathermy machine showing cutting, coagulation and bipolar energy sources. Cutting and coagulation modes are plugged in

monopolar device can be used in *cutting* and *coagulation* mode (Fig. 20.7).

- i. In **monopolar device**, the circuit is composed of a generator, active electrode, patient and patient return electrode. Thus, patient's body becomes part of the circuit when the system is activated.

The active electrode (cautery lead) is designed to be small to generate heat effectively and returning electrode (grounding pad) is designed to be large to disperse energy and prevent burn injury to the patient. The grounding pad must be large and placed securely on the patient's calf region to prevent thermal burns. It is also important that no other part of the patient should be touched by a metal (e.g. ring, bangles, etc.). Moreover, if the glove of operating surgeon is punctured and active electrode is not insulated properly, the current may find its way back through the surgeon's finger causing thermal burns to operating surgeon.

If inflammable gases are used for general anesthesia through endotracheal tube (e.g. ether; not used these days), diathermy in oral cavity should be avoided to prevent risk of explosion from electric spark.

The heat generated depends on various factors (Box 20.2).

In **cutting mode**, the monopolar device is activated in continuous waveform. It generates large amount of heat quickly over the target with

Box 20.2: Factors affecting amount of heat generated during diathermy

- Size of contact area
- Frequency of the current (power)
- Length of activation time
- Continuous or intermittent waveform

minimal lateral thermal spread. Thus, it cuts through the tissue without coagulating the vessels.

In **coagulation mode**, the monopolar device is activated in intermittent waveform. It generates less heat on a slower frequency with potential for large lateral thermal spread. This results in tissue dehydration and vessel thrombosis. For coagulation, the electrode may be applied directly or indirectly via a hemostat or tissue forceps to the bleeding tissue. The area surrounding the point to be coagulated must be dry and the controlling forceps should hold only the bleeding vessel and not the adjoining fat. Active electrode should not touch the adjoining skin as this may produce a skin burn (Box 20.3).

Box 20.3: Practical tips in diathermy use

- Large grounding pad under the patient.
- Remove all metal objects (ornaments) from the patient.
- Surgeon's gloves should not be punctured.
- Hold only the vessel that needs coagulation.
- Mop the field dry before coagulation.
- During diathermy, active electrode should not touch adjoining skin.
- Don't use diathermy near volatile gases.

A **blend waveform** can be chosen that has property of both cutting and coagulation waveform and hence can be used for simultaneous cutting as well as coagulating the tissues (Figs 20.8 and 20.9).

- ii. In **bipolar device**, a short circuit is established between the tips of the bipolar forceps that grasps the tissue. Thus, tissue grasped between the tips of the forceps completes the circuit and grounding pad is not required. The generated heat affects only the tissue in the circuit without any lateral

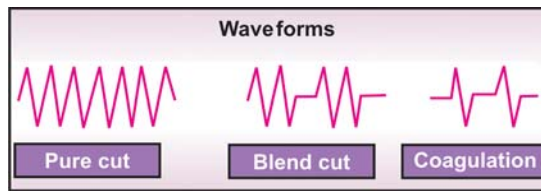


Fig. 20.8: Waveforms of diathermy



Fig. 20.9: Monopolar cautery (Blend) used for raising skin flaps during thyroidectomy

damage. Bipolar cautery is particularly useful in cauterizing vessels near vital structures like nerves, e.g. facial nerve during superficial parotidectomy, recurrent laryngeal nerve in thyroidectomy (Fig. 20.10). Moreover, bipolar cautery can also be used in wet tissues surrounded by pool of blood (unlike monopolar cautery).

Other Energy Sources Used for Tissue Cutting and Coagulation

Lasers

Laser is the acronym of Light Amplifier Stimulated Emission of Radiation.

It has two fundamental characteristics: (a) Production of identical photons by a stimulated emission process. (b) Amplification of this stimulated emission.

In laser beam, all the photons have same wavelength and same energy. The amplification of this laser beam (second characteristic) is done by passing through a "lasing medium".

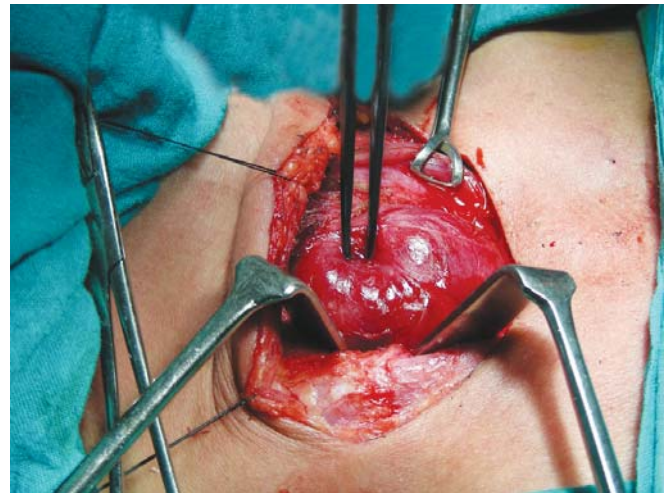


Fig. 20.10: Bipolar cautery used for achieving hemostasis near recurrent laryngeal nerve during thyroidectomy

The laser beam is used to excite molecules within the target tissue that releases energy in form of heat causing coagulation necrosis. It produces minimal collateral damage to adjoining tissues.

For practical purpose, a laser is defined by its wavelength that influences the depth of penetration of tissues (longer wavelength—deeper penetration).

The common types of lasers used in practice are:

- Argon laser:** It is absorbed by hemoglobin. Its tissue depth penetration is only 1 mm. It is used for control of bleeding from vessels of 1 mm diameter.
- CO₂ laser:** It is strongly absorbed by water. It is used for tissue vaporization (0.1 mm only) and coagulation of vessels (0.5 mm diameter only).
- Nd: YAG laser:** It is more penetrating because it is less absorbed by water as well as hemoglobin. It has tissue penetration of 10-20 mm and can coagulate vessels of 3 mm diameter.

Due to small depth of penetration, lasers have wide acceptance in dermatology and ophthalmology.

Cryosurgery

The principle of cryosurgery is that liquid nitrogen is used at extreme cold temperature (−196 degree Celsius). Its application to the tissues causes coagulation necrosis and the tissues subsequently get separated and dropped off. It is used for treatment of piles. However, there is drawback of mucus discharge and delayed pain.

High Frequency Ultrasound Waves

Ultrasound in low frequency setting causes no tissue damage and is used for diagnostic purpose. In high frequency setting, it can be used to dissect, cut and coagulate.

Harmonic Scalpel

It is an instrument that uses ultrasonic technology to dissect tissues in bipolar fashion with minimal collateral tissue damage. The device vibrates at high frequency (55000 times/sec) that generates stress and friction in the tissues leading to heat production and protein denaturation. This property helps in cutting and dissecting tissues while small blood vessels get coagulated simultaneously.

PRINCIPLES OF RADIOTHERAPY

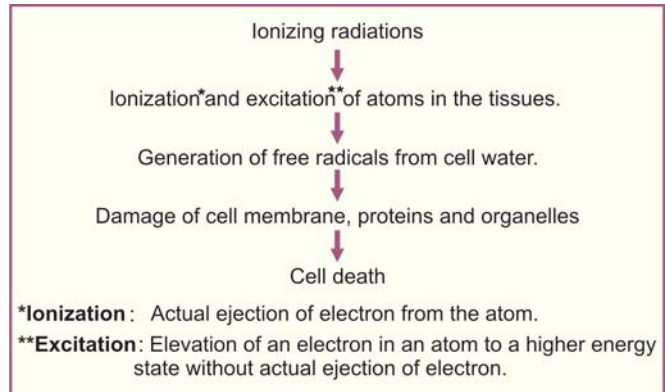
- Radiotherapy is a clinical medical speciality in which ionizing radiations are used to treat cancer and occasionally some benign diseases.
- *Aims of radiotherapy:*
 - a. In early cancer, eradication of tumor with preservation of structure and function of normal tissue.
 - b. In advanced cancer, palliation of symptoms from either the primary tumor or metastases to improve the quality of life.
- *Biological effects of radiotherapy* are shown in Box 20.4. Radiation must be able to produce double strand breaks in DNA to kill a cell since mammalian cells have a high capacity for repairing single strand damage. Tumor cells are more sensitive to lethal effects of radiation than normal tissues primarily because of difference in the ability to repair sublethal DNA damage. Presence of oxygen increases the radiosensitivity of a cell. Conversely, hypoxic cells are more resistant to radiation damage. Sulfhydryl compounds interfere with free radical generation and may act as radiation protectors.

Delivery Systems for Radiotherapy

i. Teletherapy

The beams of radiation are generated at a distance and aimed at the tumor within the patient. It is also known as *external beam radiotherapy* and is most commonly used form of radiotherapy.

Box 20.4: Biological effects of radiotherapy



The equipment is capable of generating very high energy radiation (*megavoltage*) that is precisely delivered to the tissues. Its use began with the introduction of cobalt teletherapy units but the most common source used now is the *linear accelerator (LINAC)*. Its advantages are:

- It can deliver high doses of radiotherapy to deep seated tumors.
- There is more homogenous distribution of radiation energy.
- It is skin sparing and avoids skin reactions.
- There is reduced absorption in the bones.
- There is reduced lateral scattering into adjoining tissues.

ii. Brachytherapy

The radioactive source is implanted directly into or adjacent to tumor tissue. It usually requires an operative procedure and delivers concentrated radiation doses into the tumor tissue. The doses are relatively high in comparison to doses received by the surrounding normal tissue.

If radiation source is introduced into tissues it is called *interstitial radiotherapy*. If radiation source is introduced into body cavities, it is called *intracavitary radiotherapy*.

Various radioactive isotopes used are cobalt 60, iridium 192, caesium 137 and iodine 125. These isotopes are used in custom made applicators that are flexible and highly adaptable. The isotopes are “after-loaded” under remote control into the preimplanted applicators. Its advantages are:

- Sources are introduced and removed under remote control thus preventing exposure to medical personnel.

- Position of applicator can be checked under X-rays.
- Precise dose of radiation can be delivered to the tumor tissue.

iii. Targeted Therapy

The radioactive isotope is administered systemically into the patient and it is targeted to the site of tumor, e.g. systemic administration of iodine 131 in a patient with thyroid cancer metastasis shows uptake of the isotope at site of metastasis. These days targeted therapy is being used by attaching radioisotopes to monoclonal antibodies that seek out and attach to specific tumor antigens.

Radiation Dosage (Dosimetry)

It is quantified on the basis of amount of radiation absorbed in the patient, not based upon the amount of radiation generated by the machine. Hence, its unit is called as **Rad** (Radiation absorbed dose). A Rad is 100 ergs of energy deposited per gram of tissue. A Gray (Gy) is equal to 100 rads.

$$1 \text{ cGy} = 1 \text{ rad.}$$

Radiation dose is measured by placing detectors at the body surface or calculating dose based on radiating phantoms that resemble human form.

Radiation dose has three determinants—*total absorbed dose, number of fractions and time*. For example, a typical course of radiation therapy should be described as 4500 cGy delivered to a target tissue over 5 weeks in 180 cGy fractions.

Most radiation treatment programs are delivered once a day, five days a week in 150-200 cGy fractions for 3-8 weeks.

Biologic Basis for Dose Fractionation

It is explained by the **four Rs** of radiobiology:

Repair: It represents enzymatic mechanisms for healing intracellular injury. If large dose of radiotherapy (2000 cGy) is given in a single go, it will kill both tumor as well as normal cells. However, if the same dose is given in multiple fractions (200 Gy $\times$ 10 fractions), it kills tumor cells but gives time for recovery to the normal cells.

Reoxygenation: The cells in tumor tissue become hypoxic due to overgrowth and hypoxic cells are more

radioresistant. Thus after giving radiotherapy, euoxic cells die and hypoxic cells survive. In such situation, the advantage of repeated small doses is that lethally injured euoxic cells die permitting better oxygenation of previously hypoxic cells. This process of “reoxygenation” of hypoxic cells makes them susceptible to radiotherapy during next fraction.

Repopulation: It is the ability of cell population to continue to divide and replace dying and dead cells. Thus by giving doses in fractions, it exploits the difference in recovery rate between normal tissue and tumor. The normal tissue is less damaged because recovery is slow in comparison to tumor tissue.

Redistribution: The mammalian cells have variable radiosensitivity in different phases of cell cycle. Cells in G_2 and mitosis phase are most sensitive and cells in G_0 and late S-phase are most resistant. Thus, one fraction of dose kills the cells in sensitive phase while cells in resistant phase survive. During the interval between successive dose fractions, surviving cells in resistant phase get “redistributed” to sensitive phase of the cell cycle. Thus, these cells get killed during next fraction of radiotherapy.

Thus, dose fractionation exploits the difference in **four Rs** between tumor cells and normal cells.

Types of Ionizing Radiations

Two types:

- Electromagnetic radiations (photon radiations):** X-rays and gamma rays are examples of electromagnetic radiations and these are most commonly used form of radiations used to treat cancer. Both of these cause ionization (ejection of an orbital electron) on absorption by the tissues. They differ in their source: X-rays are generated by linear accelerators, while γ -rays are generated by disintegration of atomic nuclei in radioisotopes such as cobalt and radium. Biologically, these rays behave as packets of energy called *photons*.
- Particulate radiations:** These are:
 - Electron (e)
 - Proton (p)
 - Neutron (n)
 - Alpha particle

Electrons are small, negatively charged particles. They have a very low tissue penetration and are often used

to treat superficial skin lesions (e.g. skin and lip cancers, mycosis fungoides).

Protons are positively charged particles and have a mass about 2000 times of electron.

Neutrons are not charged and have mass similar to protons. They are somewhat more effective than X-rays in treating salivary gland tumors.

Alpha particles are helium nuclei consisting of two protons and two neutrons.

However, particulate radiations have not yet found wide applicability in cancer treatment.

Clinical Applications of Radiotherapy

Pretreatment Evaluation of Patient

Before starting radiotherapy, the diagnosis should be firmly established by tissue biopsy. The disease should be staged by detailed clinical examination and relevant investigations.

Treatment Goals

Based on the type of tumor, stage of disease and condition of the patient, radiotherapy can be used in four settings:

- i. Curative
 - ii. Palliative
 - iii. Adjuvant
 - iv. Prophylactic
- i. *Curative radiotherapy*: Aim of the treatment is to eliminate all malignant cells. High doses of radiotherapy are given. It involves high cost and patient inconvenience due to long courses of treatment. There is considerable toxicity due to normal tissue damage. Curative radiotherapy has a special role in areas where preservation of anatomy and functions is of critical importance, e.g. carcinoma tongue or larynx can be destroyed by radiotherapy or removed by surgery and the chances of survival are the same. But advantage of retaining speech is worth considering with curative radiotherapy. Radiotherapy is curative for a number of malignancies (Box 20.5).
 - ii. *Palliative radiotherapy*: Aim of treatment is to control symptoms to improve the quality of life. Minimum doses of radiotherapy are given to

Box 20.5: Indications for curative radiotherapy

- Hodgkin's lymphoma
- Head and neck cancers
- Carcinoma breast
- Gynecologic cancers (cervix, ovary, uterus)
- Prostate cancer
- Carcinoma esophagus
- Carcinoma anal canal
- Testicular tumors
- Medulloblastoma
- Lung cancer (non-small cell)

Box 20.6: Indications for palliative radiotherapy

- Metastatic bone disease (for pain relief)
- Control of brain metastases
- Reversal of spinal cord compression
- Reversal of superior vena cava obstruction
- Opening of threatened airways
- Shrinkage of painful masses

achieve maximum control and minimum side effects. Short courses of treatment are given to avoid patient inconvenience and to limit the cost. Indications of palliative radiotherapy are given in Box 20.6.

- iii. *Adjuvant radiotherapy*: Radiotherapy can be combined with chemotherapy, surgery or both. Aim is to get combined benefits of different treatment modalities so as to control local as well as disseminated tumor, e.g. by giving preoperative radiotherapy in locally advanced cancer, it becomes small and less vascular, thus becoming resectable. Similarly, if surgical removal of tumor is incomplete, then postoperative radiotherapy to the surgical field helps in control of local disease. In management of carcinoma breast:
 - Surgery is for locoregional control of disease.
 - Radiotherapy is given to control any residual disease in chest wall or axilla.
 - Chemotherapy is given for systemic disease control.
- iv. *Prophylactic radiotherapy*: Certain cancers like acute leukemia and lung cancer have high incidence of developing brain metastasis even after control of primary disease. In such high-risk

settings, prophylactic cranial radiotherapy is given to prevent the occurrence of brain metastasis.

Technical Considerations

Radiotherapy is planned based on the use of a simulator with treatment fields designed to accommodate an individual patient's anatomic features. Lead shields are used to shape the field and limit the radiation exposure to normal tissues. The radiation is delivered from two or three different positions so as to deliver higher doses of radiation to target volume (tumor tissue) without increasing complications in the transit volume (normal tissue).

Toxicity

The side effects of radiotherapy are usually localized to the body site irradiated but systemic effects may also develop, e.g. fatigue, anorexia, nausea and vomiting. Organs having rapid proliferation of cells are most sensitive to radiotherapy, e.g. bone marrow, ovaries, testes, vascular endothelium and mucosal lining of intestinal tract. Organs with less renewal of cells are more resistant to radiotherapy, e.g. heart, skeletal muscles, bones and nerves.

Acute toxicity includes skin erythema and ulceration, mucositis and bone marrow depression. Most of these can be alleviated by interruption of treatment. Chronic toxicities are more serious. Various toxicities are described from head to toe in Box 20.7.

The most serious late toxicity is development of second solid tumor in or near site of radiations, e.g. development of carcinoma breast in females after chest irradiation.

PRINCIPLES OF ANESTHESIA

Introduction

On 16th October 1846, William TG Morton publicly demonstrated ether anesthesia. This marked the starting point from which Anesthesiology emerged as a specialty. **Anesthesiology** includes continuity of patient care involving preoperative evaluation, intraoperative and postoperative care and the management of systems and personnel that support

Box 20.7: Toxicity of radiotherapy

- **Head and neck irradiation**
 - Alopecia
 - Dermatitis
 - Blindness (cataract and retinal damage)
 - Mucositis, Xerostomia (dry mouth)
 - Anosmia
 - Dental caries
 - Thyroid failure
- **Bone marrow irradiation**
 - Pancytopenia, aplastic anemia
- **Chest and mediastinal irradiation**
 - Myocardial infarction
 - Constrictive pericarditis
 - Lung fibrosis
 - Spinal cord transection
 - Carcinoma breast
- **Abdomen and pelvis**
 - Enteritis (Intestines)
 - GI hemorrhage
 - Gut perforation
 - Hepatitis (Liver)
 - Nephrosclerosis (kidneys)
 - Cystitis (bladder)
 - Infertility (Testes, ovaries)
- **Fetus**
 - Chromosomal and developmental abnormalities.

these activities. Anesthesia in dentistry covers three main types of surgical procedures:

1. *Dental Chair Anesthesia*: It is outpatient anesthesia mainly for simple extraction of teeth especially in children.
2. *Day Care Anesthesia*: For extraction of molar teeth or minor oral surgery.
3. *Inpatient Anesthesia*: For complicated extraction, oral surgical procedures and maxillofacial surgical procedures.

GENERAL ANESTHESIA

General Anesthesia is defined as reversible loss of consciousness (amnesia), analgesia, neuromuscular blockade with maintenance of homeostasis.

Preanesthetic Evaluation

To formulate an anesthetic plan, thorough preanesthetic check up (PAC) should be done that includes a pertinent history, physical examination, and indicated laboratory tests. The patient is then classified according to American Society of Anesthesiologists (ASA) physical status class (Box 20.8).

Box 20.8: American Society of Anesthesiologists physical status classification

Class	Definition
1	A normal healthy patient
2	A patient with mild systemic disease and no functional limitations
3	A patient with moderate to severe systemic disease with functional limitation
4	A patient with severe systemic disease that is constant threat to life and functionally incapacitating.
5	A moribund patient who is not expected to survive 24 hours with or without surgery
6	A brain-dead patient whose organs are being harvested
E	If the procedure is an emergency, the physical status is followed by 'E'

Informed consent: No procedure should be performed without written and informed consent of the patient or guardian.

Preoperative Fasting

The goal of preoperative fasting is to decrease gastric pH and volume and subsequent aspiration of gastric contents. Currently recommended guidelines are nothing per orally (NPO) for solids 6-hours preoperatively and clear liquids up to 2-3 hours preoperatively.

Premedication

Preanesthetic medication refers to the use of drugs before anesthesia to make it more pleasant and safe. The aims are:

- Relief of anxiety
- Supplementary analgesic action
- Decreased secretions
- Antiemetic effect

- Decrease volume and acidity of gastric contents
- Amnesia.

Drugs used in preanesthetic medication are:

- Benzodiazepines (Diazepam, Lorazepam, Midazolam)
- Sedative hypnotics (Promethazine)
- Anticholinergics (Atropine, Glycopyrrolate)—decrease secretions.
- H₂ blockers (Ranitidine, Famotidine)—decrease acidity.
- Antiemetics (Metoclopramide, Ondansetron)
- Opioids (Pethidine, Morphine)

All patients with structural heart disease and prosthetic valves undergoing dental procedures associated with significant bleeding from hard or soft tissues, periodontal surgery should receive antibiotic prophylaxis (Amoxycillin) against spontaneous bacterial endocarditis (SBE).

Stages of General Anesthesia

General anesthetics cause an irregularly descending depression of central nervous system (CNS), i.e. higher functions are lost first and lower areas of brain are progressively involved.

Stage I: Stage of analgesia.

Stage II: Stage of excitement or delirium.

Stage III: Surgical anesthesia.

Stage IV: Medullary paralysis.

Inhalational Anesthetics

These are gases or vapors that diffuse rapidly across pulmonary alveoli and tissue barriers, e.g. nitrous oxide (N₂O), halothane, isoflurane, enflurane, ether, etc.

Techniques of Inhalation of Anesthetics

1. Through Anesthesia machine/apparatus
 - Open system
 - Closed system
 - Semi closed system
2. Open drop method—ether (obsolete now)

Intravenous Induction Agents

These are drugs which on intravenous injection produce loss of consciousness in one arm brain circulation time

(~11 seconds), e.g. thiopentone sodium, propofol, etomidate, methohexitone sodium, ketamine, etc. Propofol ensures clear headed recovery and has antiemetic and antipruritic properties.

Dissociative Anesthesia

It is produced by ketamine which causes profound analgesia, immobility, amnesia with light sleep and feeling of dissociation from one's own body and surroundings. Ketamine functionally dissociates the thalamus from the limbic cortex.

Neurolept Analgesia

It is produced by intravenous administration of fentanyl (opioid) and droperidol (neurolept agent) which cause general quiescence, psychic indifference and intense analgesia without unconsciousness.

Airway Management

It is an important component of anesthesia and every anesthesiologist should be highly skilled in it.

Equipment

Oral and Nasal Airways

They create an air passage between tongue and posterior pharyngeal wall (Figs 20.11A and B). Loss of upper airway muscle tone in anesthetized patients causes tongue and epiglottis to fall back against the posterior wall of the pharynx.

Face Masks

Adult and pediatric (Rendell-Baker-Soucek pediatric facemask) (Fig. 20.12).

Laryngeal Mask Airway (LMA)

It provides an alternative to ventilation through facemask on endotracheal tube. It partially protects larynx from pharyngeal secretions (but not gastric regurgitation) and it should remain in place till patient has regained airway reflex (Fig. 20.13).

Endotracheal Tubes (ETT)

These deliver anesthetic gases directly into the trachea. Most adult ETT have a cuff inflation system that creates

a seal preventing wasted ventilation and aspiration of regurgitated contents and permits positive pressure ventilation.

Rigid Laryngoscope

It permits direct laryngoscopy and intubation of trachea. It has got a handle and a blade with light bulb.

Flexible Fiberoptic Bronchoscope

It is useful in difficult airway management but requires skill.

Tracheal Intubation

Position of Patient's Head

Sniffing position: It involves flexion of cervical spine (by resting head on a pillow) and extension of atlanto-occipital joint. This position aligns oral, pharyngeal and laryngeal axes and facilitates laryngoscopy and tracheal intubation (Fig. 20.14).

Routes of Tracheal Intubation

1. *Orotracheal intubation:* It is most commonly and routinely performed route of tracheal intubation. Laryngoscope is held in left hand (non-dominant hand) and patient's mouth is opened with right hand. The blade of laryngoscope is introduced into the right side of oropharynx and tongue is swept to the left. The tip of convex blade is introduced into vallecula and straight blade (Miller) up to epiglottis. Vocal cords are visualized and tracheal tube (held in right hand) is introduced through the vocal cords into the trachea (Fig. 20.15). Cuff is inflated and proper placement of tracheal tube is confirmed by auscultation of chest and capnography. The tracheal tube is fixed *in situ* with tapes and ventilation started.
2. *Nasotracheal intubation:* Endotracheal tube is advanced through the nose into the oropharynx and guided into the trachea under direct laryngoscopic view or fiberoptic scope guided or blindly. To avoid trauma to nasal mucosa, vasoconstrictor drops (oxymetazoline) are instilled in the nostrils and water soluble jelly applied.
3. *Retromolar intubation:* It is indicated in patients with maxillo-facial trauma. Orally placed tracheal tube is positioned in the retromolar space to allow intraoperative maxillo-mandibular fixation.

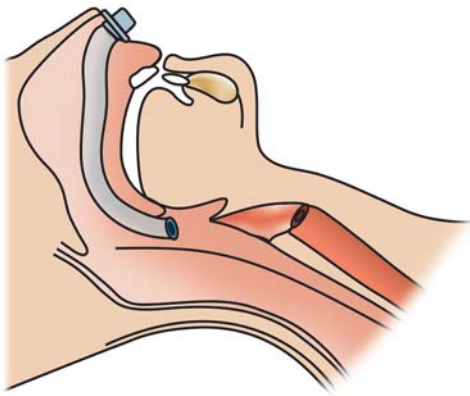


Fig. 20.11A: Nasopharyngeal airway



Fig. 20.13: Laryngeal mask airway (LMA)

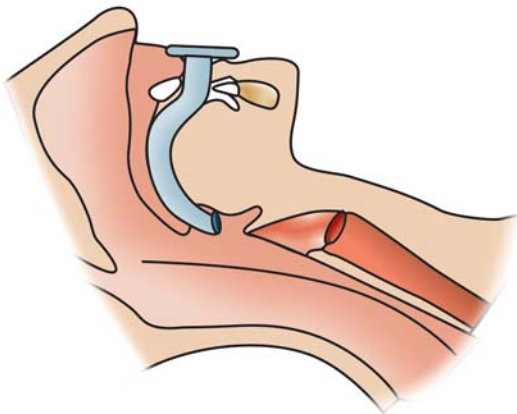


Fig. 20.11B: Oropharyngeal airway



Fig. 20.14: Direct laryngoscopy



Fig. 20.12: Mask ventilation

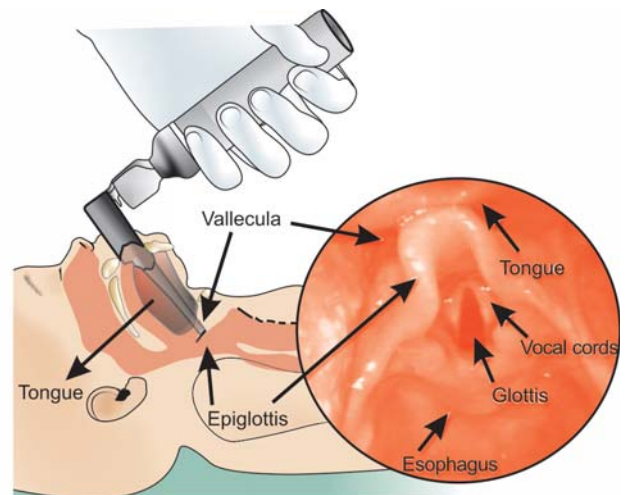


Fig. 20.15: Structures visualized on direct laryngoscopy at the time of endotracheal intubation

4. *Submento-tracheal intubation*: It is indicated in patients with maxillo-facial trauma when retromolar intubation is not possible. Orally placed tracheal tube is brought out in the submental region by an incision and blunt dissection through mylohyoid muscle.
5. *Surgical airway (Tracheostomy)*: Rarely indicated for perioperative airway management.

Neuromuscular Blockers

Neuromuscular blocking drugs cause skeletal muscle relaxation. The term 'muscle relaxants' should never be used for neuromuscular blockers. Neuromuscular blockers are of two types:

- i. *Depolarizing neuromuscular blockers*: Succinylcholine, decamethonium. These agents act like acetylcholine receptor agonists and cause continuous end plate depolarization resulting in blockade of neuromuscular transmission and thus muscle relaxation.

Depolarizing neuromuscular blockers are metabolized in plasma and liver by enzyme pseudo cholinesterase. Succinylcholine is preferred agent for rapid sequence intubation. The block produced by succinylcholine is characterized by visible motor unit contractions called fasciculation.

- ii. *Non-depolarizing neuromuscular blockers*: They are further of three types
 - a. Long acting: Pancuronium, tubocurarine, gallamine
 - b. Intermediate acting: Atracurium, vecuronium, rocuronium
 - c. Short acting: Mivacurium

Rocuronium is best among non-depolarizing agents for rapid sequence induction. Atracurium is preferred agent in patients with renal or liver disorders and vecuronium in patients with cardiac disease. Non-depolarizing neuromuscular blockers are competitive antagonists of acetylcholine receptors. They are not metabolized by pseudo-cholinesterase. Reversal of non-depolarizing neuromuscular blockade requires administration of reversal agents (cholinesterase inhibitors) which inhibit metabolism of acetylcholine.

Analgesia

Pain relief is right of every patient and it should be provided to all.

Intraoperative Analgesia

Opioids are the commonly used agents like fentanyl, sufentanil, morphine, pethidine and tramadol. If ketamine is used for induction of anesthesia, it also offers profound analgesia.

Postoperative Analgesia

It is provided by tramadol, diclofenac, ketorolac, epidural bupivacaine alone or bupivacaine plus opioids (fentanyl, tramadol, morphine). Diclofenac patch offers the advantage of avoiding injections. In pediatric patients, paracetamol or NSAIDs syrup and suppositories of paracetamol and diclofenac are commonly used.

Monitoring

Intraoperative monitoring includes noninvasive blood pressure, heart rate, ECG, peripheral arterial oxygen saturation (SpO₂), color of mucosa, temperature, precordial and esophageal stethoscope, end tidal carbon dioxide, anesthetic gas analysis, neuromuscular monitors, intravenous fluids input, urine output and blood loss. If indicated, more invasive monitoring is done (in cardiac surgery, major surgical procedures): invasive blood pressure, central venous pressure (CVP), pulmonary artery pressure (PAP), pulmonary capillary wedge pressure (PCWP), continuous cardiac output (CCO), cardiac index (CI), etc. One of the primary responsibilities of an anesthesiologist is to act as a guardian for anesthetized patient during perioperative period and be always vigilant.

Extubation of Trachea

Extubation of trachea is performed when patient is either awake or deeply anesthetized with adequate respiration. Residual neuromuscular blockade is reversed by administration of *cholinesterase inhibitors* (neostigmine) and *anti-cholinergic agents* (glycopyrrolate or atropine). Cholinesterase inhibition increases the concentration of acetylcholine, thereby re-establishing neuromuscular transmission. Thorough oropharyngeal suctioning is done, cuff of tracheal tube deflated and tracheal tube removed gently and 100% oxygen is administered by face mask for few minutes to avoid *diffusion hypoxia*.

LOCAL ANESTHESIA

With advances in medications and techniques, clinical dentistry has evolved from its image as a “painful experience” to a clinically “painless service”. In this direction, local anesthesia is the backbone of contemporary dental and oral surgical practice.

Mechanism of Action

Local anesthetics are drugs that reversibly block the generation and propagation of impulses in excitable tissue. Local anesthetic agents cause direct inhibition of voltage gated sodium channels which prevents influx of sodium across the neuronal cell membrane. Sodium ion influx is required for initiation and propagation of action potential.

Pharmacological Effects of Local Anesthetics

The major advantage of local anesthetic actions is its reversibility with no evidence of damage to nerve fibers or cells.

1. *Local*: Nerve blockade and direct effect on smooth muscle.
2. *Regional*: Loss of pain, temperature, touch sensation; loss of motor power and vasomotor tone.
3. *Systemic*:
 - a. CVS—depress myocardial automaticity, contractility and conduction velocity. Local anesthetics are anti-arrhythmics but in high doses can induce arrhythmias.
 - b. Respiratory system—depress hypoxic drive, produce bronchial smooth muscle relaxation.
 - c. CNS—neuronal inhibition, in high doses can cause convulsions.

Chemical Structure and Classification

The typical local anesthetic molecule (lidocaine as prototype) contains a tertiary amine attached to a substituted aromatic ring by an intermediate chain. The intermediate chain contains either an ester or an amide linkage. The *local anesthetics are classified as*:

1. *Aminoesters*: Cocaine, procaine, chlorprocaine, tetracaine, benzocaine
2. *Aminoamides*: Lidocaine, bupivacaine, dibucaine, ropivacaine, prilocaine.

Aminoester local anesthetics are generally less effective than amides because they have poor diffusion properties. Also, procaine has significant allergenicity.

Preparations of Local Anesthetics

Local anesthetics containing epinephrine are acidic (pH, 5.0) to inhibit oxidation of epinephrine. pH of local anesthetics without epinephrine is 5.5 to 7.00. Retardation of oxidation to increase shelf-life is also achieved by addition of antioxidant sodium bisulfite in 0.05-1% concentration, which lowers the pH of solution. Antimicrobials most commonly used in commercial preparations are methyl paraben, ethyl paraben and propyl paraben. They are potent allergens and have been implicated in allergic reactions initially attributed to local anesthetics.

Local Distribution

Intraneural injection of local anesthetic is painful and may result in nerve damage. Thus, local anesthetics are always injected near the nerve in a minimum volume and a minimum concentration.

Clearance

Aminoamides are primarily metabolized in liver and aminoester local anesthetics are cleared by plasma and liver cholinesterase enzyme.

Indications of Local Anesthesia in Dentistry

1. Extraction of teeth
2. Odontectomy or surgical removal of teeth
3. Alveoloplasty and alveolectomy
4. Incision and drainage of abscess
5. Cavity preparation
6. Pulpectomy, pulpotomy
7. Cyst enucleation
8. Peridontal and gingival procedures
9. Relief of sore spots
10. Treatment of trismus
11. Removal of small neoplastic growths and salivary stones
12. Diagnosis and treatment of various orofacial pains
13. For anesthesia of oral cavity and jaw bones for routine surgical procedures, like treatment of fractures, growth, etc.
14. In radiography, when patient is gagging due to placement of film in mouth

Advantages of Local Anesthesia

- Patient is awake and conscious.

- No need for pre-procedure fasting.
- Very low morbidity.
- No requirement of trained anesthesiologists and other personnel.
- Easy to administer, with low failure rates.
- Patient can leave the dental office after the procedure unescorted.

Contraindications

- Local anesthesia is not recommended in fearful and apprehensive patients.
- Allergy to local anesthetic solution
- Acute infection
- Mentally challenged and uncooperative children
- Major oral surgical procedures.

In such conditions, general anesthesia is indicated.

Technique

Requirements: Disposable syringes, disposable needles, local anesthetic solutions and cartridges, cleaning solutions.

- Topical or surface anesthesia:** It is effective on the mucous membrane. The onset of anesthesia is immediate but effect is of short duration. There is no requirement of any injection.
- Infiltration anesthesia:** subcutaneous, submucosal, subperiosteal, intraosseous, pericemental, intrapulpal.
- Block (conduction) anesthesia:** Inferior alveolar nerve (dental nerve), mental nerve, infraorbital nerve, posterior superior alveolar nerve (Fig. 20.16).

Local Anesthetic Agents

Routinely used agents for local anesthesia are:

i. Lidocaine

It is a prototype dental local anesthetic. It has excellent anesthetic efficacy with harmful allergenicity. It is available as plain lidocaine 2% or lidocaine 2% with epinephrine (1 : 50,000-1 : 2,00,000). Maximum dose for plain lidocaine 2% is 3 mg/kg and for lidocaine 2% with epinephrine (1 : 2,00,000) is 7 mg/kg. Lidocaine 2% with epinephrine rapidly induces anesthesia that lasts for 80-90 minutes.

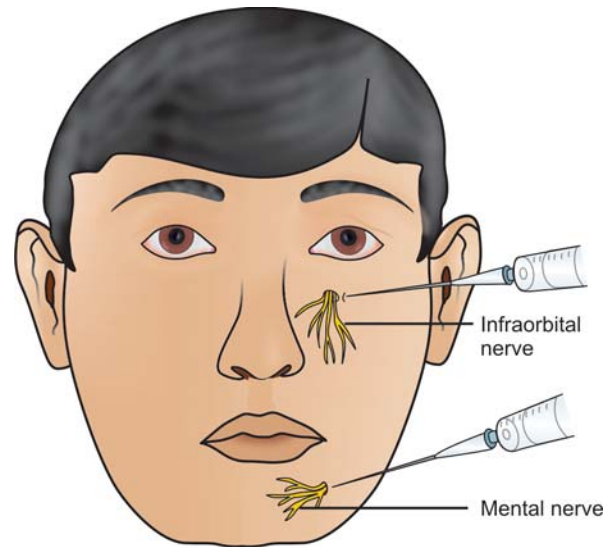


Fig. 20.16: Sites of nerve block on face

Lidocaine is also available as lidocaine 5% ointment, lidocaine 2% jelly, lidocaine 5% viscous, lidocaine 10% aerosol spray, lidocaine 4% for topical anesthesia.

Amount of local anesthetic to be administered is related to patient's age and weight. In children, the amount to be injected is reduced according to the weight and/or age:

Young's Rule: Child's dose = Child's age / Age + 12

Clark's Rule: Child's dose = Child's weight (in pounds)/150

ii. Bupivacaine

It has longer onset time and longer duration of action (4-5 hours). It is available as 0.5% bupivacaine plain, 0.5% bupivacaine with 1 : 2,00,000 adrenaline. The maximum dose is 2 mg/kg.

Complications of Local Anesthesia

Local Complications

Pain at site of injection, burning sensation on injection, hematoma formation, intravascular injection, diplopia, transient blindness, needle breakage.

Systemic Complications

Acute circulatory insufficiency (orthostatic hypotension), toxic reactions (arrhythmias, hypotension, convulsions,

cardiac arrest), allergy to local anesthetic/epinephrine/methyl paraben, hyperventilation tetany.

Late Complications

Infection, necrosis, trismus, prolonged anesthesia/paresthesia, post-injection herpes.

Management of Overdose Reaction

Symptomatic treatment is given to the patient. Maintain airway, breathing and circulation. Oxygen therapy is instituted. Anticonvulsants and ventilatory support is given, if required. If allergic reaction occurs, it is managed by administration of antihistaminic agent diphenhydramine 25-50 mg and hydrocortisone 100 mg intravenously. If required, intravenous bronchodilators and epinephrine (intravenous/nebulization/subcutaneous) can be administered.

CENTRAL NEURAXIAL BLOCKS

Spinal, epidural and caudal blocks are known as central blocks because they involve injection of local anesthetics in to or immediately adjacent to the spinal cord.

Spinal Anesthesia

Injection of local anesthetics into the subarachnoid space produces spinal anesthesia. It is *indicated* in procedures

involving lower extremities, hip, perineum and lower abdomen. The *contraindications* of administering spinal anesthesia are refusal by the patient, skin infection at lumbar puncture site, severe hypovolemia, coagulopathy and increased intracranial pressure.

Technique

The spinal needle is introduced in L3-L4 or L2-L3 interspace (below L 1, as puncture of intact spinal cord is less likely) and it pierces skin, subcutaneous tissue, supraspinous and interspinous ligaments, ligamentum flavum, dura mater and arachnoid mater. Once there is clear free flow of cerebrospinal fluid, local anesthetic agent is injected.

The block produced by spinal anesthesia is sympathetic blockade (judged by temperature sensation) and is two segments higher than sensory block (pain, light touch) that in turn is two segments higher than motor blockade. Spinal anesthesia produces total sympathetic block resulting in increase in volume of capacitance vessels, subsequent decrease in venous return to heart and hypotension. To treat hypotension, I/V fluids, mephentermine and ephedrine are commonly used.

21

Chapter

Fractures and Maxillofacial Fractures

Sanjay Marwah, Virendra Singh

DEFINITIONS

Fracture

It is the structural break in normal continuity of bone.

Dislocation

It is a complete disruption of a joint with no remaining contact between articular surfaces.

Subluxation

It is a partial disruption of a joint with some contact remaining between articular surfaces.

Sprain

It is a painful condition due to tearing of a ligament and soft tissue injury.

TYPES OF BONES

Tubular Bones

These are long bones with marrow in the medullary canal, e.g. femur.

Cancellous Bones

These are flat bones that have uniform spongy texture with no medullary canal, e.g. sternum.

TYPES OF FRACTURES

Simple Fracture

A fracture is called as simple or closed when there is no communication between site of fracture and exterior of body.

Compound Fracture

A fracture is called compound or open when there is a wound on the skin surface leading down to the site of fracture. However, it must be stressed that the presence of a skin wound and fracture of underlying bone without any communication between the two is not a compound fracture.

In compound fracture, there is a risk of contamination of fractured bone by outside organisms while a closed fracture is free from this risk.

CLASSIFICATION OF FRACTURES

Classification Based on Etiology of Fractures

Traumatic fracture: It forms the largest group and the term 'fracture' generally means traumatic fracture.

It occurs in bones with normal strength. It may be caused by *direct violence*, e.g. fracture mandible due to blow on face or by *indirect violence*, e.g. condylar fracture due to trauma over chin region.

Stress fracture (Fatigue fracture): It occurs due to repeated injury occurring at the same site.

It occurs in bones with normal strength. The mechanical structure of the bone gets fatigued due to repeated trauma and then bone breaks, e.g. fracture second metatarsal bone due to prolonged marching in soldiers (**march fracture**).

Pathological fracture: It occurs in a bone already weakened by disease. The bone gets fractured due to trivial injury or even spontaneously. The causes of pathological fracture are given in Box 21.1.

Box 21.1: Causes of pathological fracture

Local Diseases of Bone	
Infections	Pyogenic osteomyelitis
Benign tumors	Osteoclastoma
Malignant tumors	Osteogenic sarcoma
	Ewing's tumor
	Metastatic carcinoma (from breast, lung, thyroid)
Miscellaneous	Simple bone cyst
	Bone atrophy (in polio)
	Tabes dorsalis
Generalized Diseases of Bone	
Congenital	Osteogenesis imperfecta
Diffuse rarefaction of bone	Hyperparathyroidism
	Senile osteoporosis
	Rickets
	Osteomalacia
Disseminated tumors	Multiple myeloma
Miscellaneous	Paget's disease
	Fibrous dysplasia

Classification of Patterns of Fracture (Fig. 21.1)

Transverse fracture: It is due to bending of bone along its long axis. It is unlikely to become redisplaced after reduction.

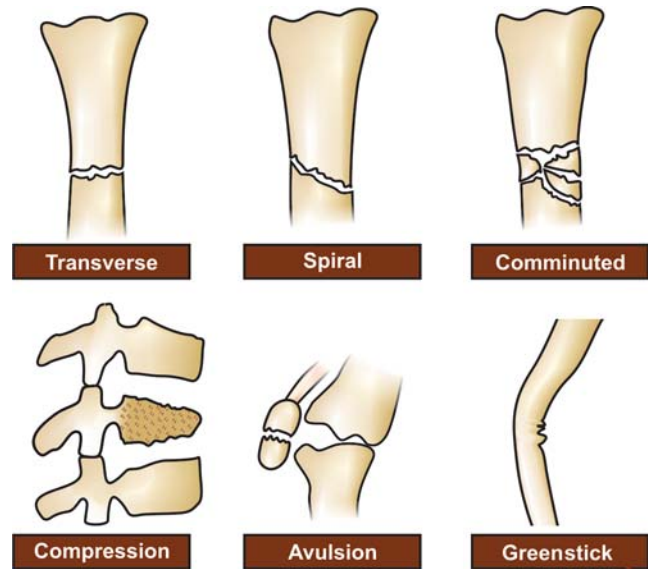
Spiral fracture: It is caused by twisting of long bone along its axis. It is prone to redisplacement after reduction.

Comminuted fracture: It is due to severe injury that breaks the bone into fragments.

Compression fracture: It is caused by force applied along the length of a bone and the bone collapses into itself, e.g. compression fracture of vertebral body due to fall from a height. As the spongy bone is crushed so it cannot be restored to its original form.

Avulsion fracture: It is caused by severe traction on a ligament that breaks the bone on which it is inserted. It is commonly seen in small bones attached with strong muscles, e.g. patella (attached to quadriceps muscle).

Greenstick fracture: It is seen in children whose bones are flexible. An angulation force bends the bone at one

**Fig. 21.1: Patterns of fracture**

cortex and breaks it at the other thus producing an incomplete fracture.

HEALING OF A FRACTURE

As soon as the bone breaks, the fracture begins to heal. Various stages in healing of fracture in a tubular bone are (Fig. 21.2A):

Stage of hematoma: The torn vessels form a hematoma between and around the fracture surfaces. The ring of bone immediately adjacent to each side of the fracture becomes ischemic and undergoes necrosis.

Stage of subperiosteal and endosteal cellular proliferation: These cells are precursors of osteoblasts. They form a collar of active tissue that grows towards the other fragment. The blood clot is pushed aside by the proliferating tissue and gets absorbed.

Stage of callus: The proliferating cells give rise to osteoblasts that form the immature woven bone of fracture callus. This mass of callus is visible in radiographs and can be felt as a hard mass surrounding the fracture site in superficial bones.

Stage of consolidation: The woven bone gradually transforms into mature bone that has typical lamellar structure.

Stage of remodeling: The bone is gradually strengthened along the lines of stress and surplus bone is resorbed

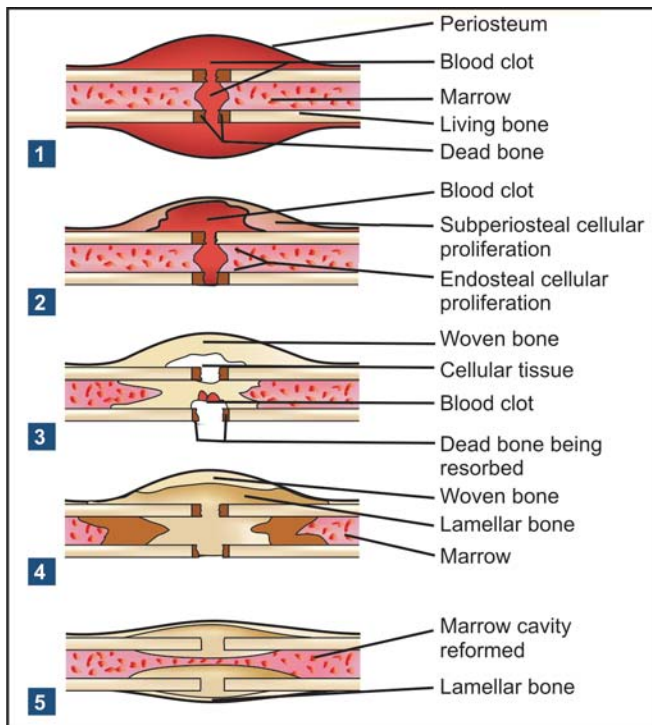


Fig. 21.2A: Stages in healing of a fracture

outside the lines of stress. Thus, the bone is restored to more or less of its original form.

In *cancellous bone*, as the bone has uniform spongy texture and no medullary canal, there is broad area of contact at fracture site. So healing occurs without medium of callus. However, pathological events are similar to that of fracture tubular bone.

CLINICAL FEATURES AND DIAGNOSIS

History

- Mostly there is history of injury except in pathological or stress fracture.
- The patient complains of pain at site of fracture.
- There is loss of function in the injured area, e.g. in limb fracture, patient is reluctant to move it.
- The patient may complain of weakness in the limb or loss of sensation due to neurological damage.

Examination

- Swelling and bruising at the site of injury.
- There may be external wound suggesting compound fracture.

- Localized tenderness at the site of fracture.
- Local temperature is raised due to inflammatory response.
- On limb movement, abnormal mobility or crepitation may be elicited. However, vigorous efforts should not be made to elicit this sign as it causes severe pain and further soft tissue damage and blood loss.
- Examine for neurovascular damage in the injured limb by checking distal circulation and any neurological deficit.

Radiological Examination

- The X-ray should include the whole bone including the joint above and below.
- X-rays should be taken in two planes at right angle to each other (anteroposterior and lateral).
- Sometimes oblique view is also required to detect fracture.
- The information provided by X-ray is shown in Box 21.2.

Box 21.2: Information provided by X-ray

- Accurate localization of fracture site.
- Demonstrates dislocation if any.
- Demonstrates degree and direction of displacement.
- Provides evidence of underlying bone pathology.
- It may show a radiopaque foreign body.
- It may reveal an unsuspected injury.

MANAGEMENT

First Aid

At the site of accident, the aim of management is to keep the patient alive and to minimize the chances of further damage. The measures include:

- Maintenance of adequate airway and breathing.
- Maintenance of circulation by control of bleeding. The external bleeding is controlled by application of pressure dressing (using cloth, bandage, handkerchief or manual pressure).
- The use of tourniquet should be avoided as it may only impair venous return causing increased bleeding. Moreover, if it is kept for too long, it may cause ischemic limb damage.
- The limb should be splinted with whatever method is available (piece of wood, plastic, umbrella, etc.).

- If spinal injury is suspected, the patient should be moved without rotating and flexing the spine (log roll).

Definitive Management

It is done in the hospital. It has two components:

General Management of the Patient

- Treatment of shock due to blood loss initially by intravenous crystalloids and colloids followed by blood transfusion.
- Pain control by parenteral analgesics (diclofenac sodium, tramadol).
- Broad spectrum antibiotics are given parenterally especially in compound fractures to prevent wound infection.
- Prophylaxis against tetanus with tetanus toxoid injection.
- Management of associated injuries.

Local Management of the Fracture

The aims of local treatment of fracture are:

- Pain relief.
- Reduction of fracture.
- Immobilization to promote fracture healing.
- Preservation and restoration of function.

Treatment varies from individual to individual and should not be guided by X-ray findings alone. The aim is 'to treat the patient, not the radiograph'.

Treatment of Uncomplicated Closed Fractures

The treatment includes:

Reduction

Fracture reduction is done only if it is necessary. Not all fractures need reduction even if displaced because there is no change in final outcome. The reduction is done only if there is difficulty in union or risk of impairment in functions. The reduction can be achieved by:

- Closed manipulation under anesthesia:* The fragments are grasped, disimpacted and then adjusted to near normal position.
- Reduction by mechanical traction:* The traction is applied by weights (Fig. 21.2B).
- Operative reduction:* During operation, the fragments are reduced under vision and fixed internally to maintain the position.

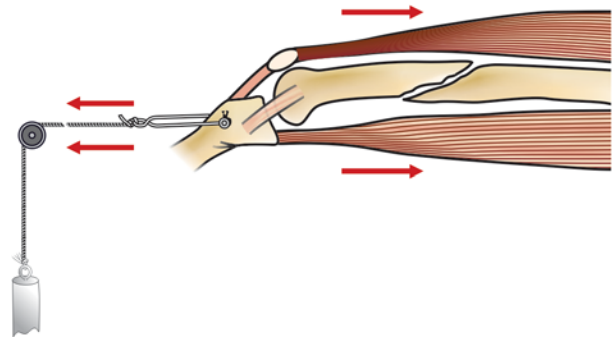


Fig. 21.2B: Continuous skeletal traction with a hanging weight counteracts muscle pull and prevents overlap of bone fragments

Immobilization

The aims of immobilization are:

- To prevent movement
- To prevent displacement
- To relieve pain.

The methods of immobilization are:

- Plaster of Paris (POP) cast or splint*
- Immobilization by continuous traction:* It is required in spiral fracture to prevent overlap of the fragments due to muscle pull (Fig. 21.2B).
- Immobilization by internal fixation:* It is done when POP cast or traction is unable to give immobilization. Also, it is used in case fracture requires open reduction. For internal fixation, the bone on either side of fracture site is exposed by dissecting soft tissues and immobilization is achieved by one of the following ways:
 - Plate held with screws (Fig. 21.2C).
 - Transfixation screws (Fig. 21.2D).
 - Intra-medullary nail (Fig. 21.2E)
 - Circumferential wires (Fig. 21.2F).
- Immobilization by external fixation:* It is done in case of open fracture (see below).

Rehabilitation

The results of fracture treatment are significantly improved by rehabilitation. It should begin as soon as treatment of fracture starts.

The prolonged rest in an injured limb can lead to collection of edema fluid around fracture as well as in the whole limb. Also there is muscle wasting and joint stiffness.

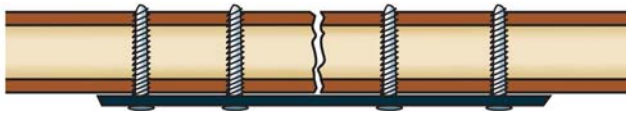


Fig. 21.2C: Plate held with screws



Fig. 21.2D: Transfixation screws



Fig. 21.2E: Intramedullary nail



Fig. 21.2F: Circumferential wires

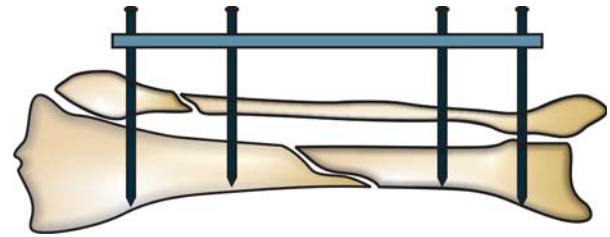


Fig. 21.2G: External fixation

The aims of rehabilitation are:

- To preserve functions while fracture is uniting.
- To restore functions after fracture is united.

The two essential methods of rehabilitation are: **active use** and **active exercises**.

Active use implies that the patient should continue to use the injured part as naturally as possible. Although rest is necessary in early days following injury, but the injured part should gradually return to activity as soon as possible.

Active exercises imply doing exercises of muscles and joints under supervision of a physiotherapist. It should be encouraged at an early stage. In case a limb is immobilized in a splint, muscle functions are preserved by static contraction of muscles without moving the joint. When splints are no longer required, active joint movements are started.

When a fracture has soundly united, physiotherapy is intensified by carrying movements against gradually increasing resistance until normal power is regained.

Treatment of Open Fractures

The open fracture demands urgent attention so as to minimize the risk of wound infection. The principles of treatment are:

- Wound debridement:** All extraneous material is removed. The dead and devitalized tissue is excised leaving healthy and vascularized tissue.
- If wound is clean and is dealt within few hours of injury, it should be closed primarily.
- In case of dirty, severely contaminated wound with delayed presentation (more than 8-10 hrs), it should be left open and dressed regularly. Once wound becomes clean, delayed closure is done.
- Treatment of fracture:** Principles of management are same as for closed fractures. However, open reduction and internal fixation of the fracture should be avoided to prevent the risk of infection. If fracture is unstable and unsuitable for treatment by plaster of Paris alone, **external fixation** by pins inserted into the bone fragments and fixed to a rigid external bar should be done (Fig. 21.2G).

NEWER METHODS OF FRACTURE TREATMENT

The following methods of fracture treatment have come up recently and these methods have revolutionized the treatment of fractures.

- Biological fixation of fractures:** The internal fixation of fracture is done without opening periosteum and minimal disruption of soft tissues at fracture site. Thus, fracture hematoma is not disturbed and osteogenic potential at fracture site is maintained that helps in early union of fracture.
- Use of image intensifier:** It is an X-ray screening device used on operation table at the time of fracture reduction. It helps in accurate fracture reduction and precise positioning of implants that is confirmed on operation table itself. Its use has revolutionized the fracture treatment. Earlier check X-rays were done after surgery in radiology suite and any improper fracture reduction required re-exploration.

- c. *Biodegradable implants*: The plates and screws are made of biodegradable polymers which provide strength for adequate period of time and then get metabolized within the body. Thus, long-term complications of metal implants are avoided.
- d. *Arthroscopic (Key hole) surgery*: In joint injuries, endoscopes are used to visualize the site of fracture that can be reduced precisely. The tears in ligaments and cartilage are also taken care of.
- e. *Video assisted surgery*: In spinal injuries, video assisted minimal invasive surgery is performed for passing screws through fracture site for stabilization. If performed with robots, it makes the surgery precise without causing damage to adjoining nerves and spinal cord (See Chapter 1: Introduction to Surgery).
- f. *Light weight plaster*: Instead of plaster of Paris (POP), PVC material is used (Deltalite) for fracture immobilization. Its advantages are that it is 1/3rd of the weight of POP, 10 times stronger than POP, porous and comfortable.

COMPLICATIONS OF FRACTURES

These can be divided into two groups:

- i. Complications related to fracture itself:
 - Infection: Osteomyelitis, tetanus and gas gangrene.
 - Delayed union
 - Nonunion
 - Malunion
 - Shortening
 - Avascular necrosis
- ii. Complications due to associated injuries:
 - Injury to blood vessels
 - Injury to nerves
 - Injury to tendons
 - Injury to joints
 - Injury to adjoining viscera
 - Fat embolism
 - Deep vein thrombosis and pulmonary embolism.

FRACTURES OF HEAD AND NECK REGION

These include:

- *Skull fractures* (see Chapter 17: Nervous System).
- *Fracture cervical spine* (see Chapter 10: Care of the Acutely Injured).

• Maxillofacial fractures

Causes are:

- Road traffic accidents
- Domestic violence
- Sports injuries.

MAXILLOFACIAL FRACTURES— CLASSIFICATION

Fractures of the facial skeleton can be divided into three parts:

Upper third: It involves area of the skull above eye brows. The fractures usually involve frontal sinuses and supraorbital ridges.

Middle third: It involves area between eyebrows and mouth. The bones fractured are maxilla, zygomatic complex and nasal bone.

Lower third: It involves fractures of the mandible.

The fractures tend to occur through weak areas like sutures, foramina and thin bony parts.

Another way of dividing fractures of facial skeleton is:

- I. Fractures not involving dental occlusion (nose, zygomatic bone).
- II. Fractures involving dental occlusion (maxilla and mandible).

This division is based on the fact that provision and maintenance of proper dental occlusion is the mainstay of treatment of facial fractures.

EARLY CARE

- Majority of patients with maxillofacial fractures require temporary splinting by passing through a wire around the teeth adjacent to fracture line (bridal wiring).
- In case of Le Fort fracture with palatal split, trans-palatal wiring is an essential step to stop the nasal bleeding along with nasal packing.
- Splinting of bilateral parasymphysis fracture prevents tongue from falling back and thereby restores the compromised air way.
- In case of unstable fracture mandible, support of barrel bandage may be used to support the mandible.
- In cases of polytrauma having multiple facial lacerations, multiple fractures of facial skeleton and head injury, there is risk of:

- ☐ Immediate or delayed respiratory obstruction.
- ☐ Severe uncontrolled facial hemorrhage.

The patient should be nursed in semiprone position so that bleeding and secretions fall out with gravity and aspiration is prevented. The detailed management is given in Chapter 10: Care of the Acutely Injured.

CLINICAL EXAMINATION

- Primary survey of the patient is done as per ATLS guidelines. (See Chapter 10: Care of the Acutely Injured).
- Examine whole head and face visually and by palpation using gloved hands.
- Start with the facial lacerations and soft tissue injuries.
- Feel for any bony tenderness, asymmetry and step formation starting from above downwards:
 - ☐ Supraorbital and infraorbital margins.
 - ☐ Nasal bridge.
 - ☐ Zygomatic arches.
 - ☐ Maxilla.
 - ☐ Mandible.
- Swelling, change of contour of nasal bridge or a new asymmetry suggests a fractured nose (Fig. 21.3).
- Examine eyes for subconjunctival hemorrhage, diplopia and visual acuity.
- Do intraoral examination under good light.
- Examine for the dental occlusion. In case of malocclusion of teeth, a fracture of the jaw (maxilla, mandible) is suspected.



Fig. 21.3: Fracture nasal bone; see swelling and change in contour of nasal bridge

- Examine for the relevant cranial nerves for anesthesia or paresthesia.
- There can be facial nerve palsy due to injury to branches of facial nerve or due to fracture temporal bone.
- In case of tearing of gingiva with loose tooth, fracture of alveolus is suspected.
- In *fracture of the maxilla*, findings are:
 - ☐ Face as a whole, especially middle third, is diffusely swollen with edema of cheeks and eyelids that 'looks like a football'.
 - ☐ Some diagnostic signs for mid face fractures are:
 - **Gurien sign** with floating maxilla is feature of Le Fort I
 - **Battle sign** with **panda face** are features of Le Fort II and III
 - **Dish face appearance** is a feature of Le Fort III
 - ☐ CSF rhinorrhea. It differs from nasal discharge in that it does not starch the cloth whereas nasal secretions do.
 - ☐ Subconjunctival hemorrhage and black eye.
 - ☐ Orbital symptoms (diplopia, diminished vision, exophthalmos, enophthalmos).
 - ☐ Failure of occlusion of teeth.
 - ☐ Test for the mobility of maxillary complex by grasping maxilla just above incisors between finger and thumb of one hand while fingers and thumb of other hand holds the head steady at bridge of the nose (Fig. 21.4). If maxilla is fractured, gentle backward and forward movement of the fingers will reveal the mobility of maxilla.
- In *fracture of the mandible*, findings are:
 - ☐ Swelling and skin discoloration in the lower part of the face (see Fig. 16.7).
 - ☐ Hematoma of the floor of mouth (*Coleman's sign*).
 - ☐ Improper occlusion of teeth.
 - ☐ Combined intraoral and extraoral palpation reveals break in continuity of the mandible and bony crepitus.
 - ☐ In unilateral condylar fracture, there is deviation of the jaw to the affected side on opening mouth.
 - ☐ Guardsman fracture is feature of bilateral condylar fracture associated with symphysis fracture.



Fig. 21.4: Method of testing mobility of maxillary complex in fracture maxilla

- ☐ There may be anesthesia of lower lip due to inferior dental nerve damage.
- In *fracture of the zygomatic complex*, findings are:
 - ☐ Soft tissue swelling and bruising over the cheek bone.
 - ☐ Flattening of cheek prominence (Figs 21.5A and B)
 - ☐ Subconjunctival hemorrhage.
 - ☐ Fracture line may be palpable in upper buccal sulcus.
 - ☐ Anesthesia of upper lip and upper teeth due to damage of branches of infraorbital nerve.
 - ☐ Mongoloid slant—downward displacement of lateral eye brow.
 - ☐ Hypoglobus—vertically downward displacement of eye globe.
 - ☐ Diplopia due to fracture of orbital floor causing damage to sling mechanism of eyeball.

RADIOLOGICAL INVESTIGATIONS

These are performed only after stabilizing the general condition of the patient.

Following X-rays are done for different areas:

For Fracture Mandible

- a. Posteroanterior view of mandible in open mouth position: For symphysis, lower border of the body and angle of the mandible (Fig. 21.6A).



Fig. 21.5A: Zygomatic arch fracture showing flattening of cheek prominence (lateral view)



Fig. 21.5B: Zygomatic arch fracture showing flattening of right cheek prominence (frontal view)

- b. AP view (Towne's view) of mandible for head and neck of the condyle of mandible (Fig. 21.6B).
- c. Right and left lateral oblique view of mandible: For body and ramus of the mandible.
- d. Orthopantomograph: For complete mandible from condyle to condyle (Fig. 21.7).
- e. Occlusal view of the mandible to see split fractures in body of the mandible.

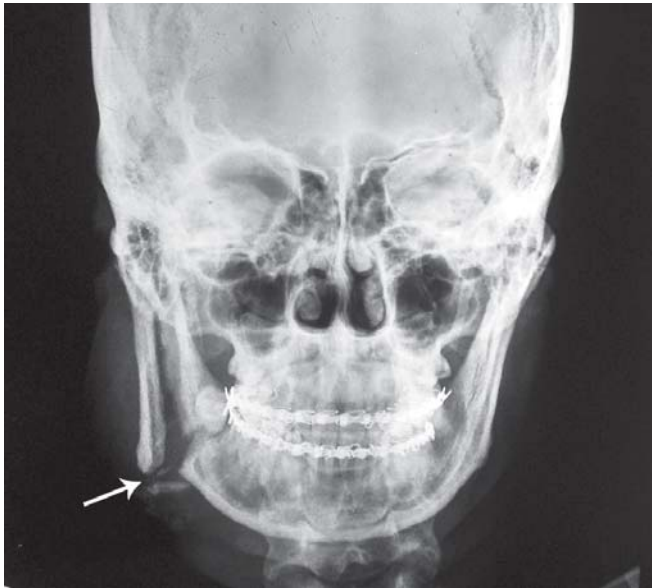


Fig. 21.6A: X-ray facial skeleton PA view showing fracture ramus and fracture angle of mandible on right side



Fig. 21.6B: Towne's view showing bilateral condylar fracture

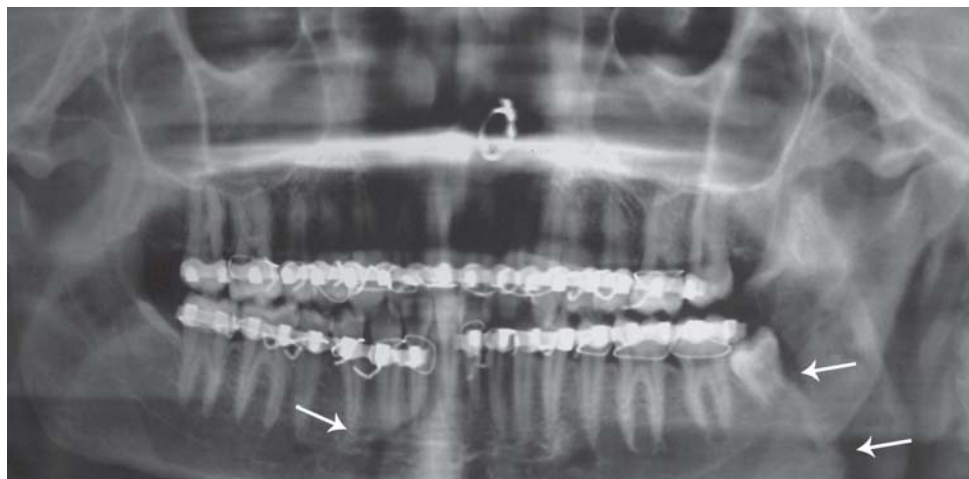


Fig. 21.7: OPG showing fracture right parasymphysis and displaced angle of mandible left side

For Fracture Maxilla

- Posteroanterior view maxilla in Water's position—it also shows zygomatic bone and infraorbital margins.
- 30° occipitomenal projection (Sinus view) (Fig. 21.8A).

For Fracture Zygomatic Arch

Superoinferior projection (Submentovertex view or Jug handle view) (Figs 21.8B and C).

For Fracture Nasal Bone

True lateral view of the skull (Fig. 21.9).

CT scan is more useful for complex maxillo-facial injuries especially middle third fractures.

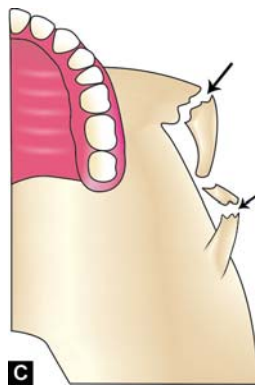
TREATMENT

General Measures

- Since all faciomaxillary fractures are likely to be compound fractures, so broad spectrum antibiotics



Fig. 21.8A: Occipitomeatal view showing fracture of right maxillary bone and left zygomatic bone



Figs 21.8B and C: Zygomatic arch fracture:
(B) Submentovertex view and (C) Line diagram

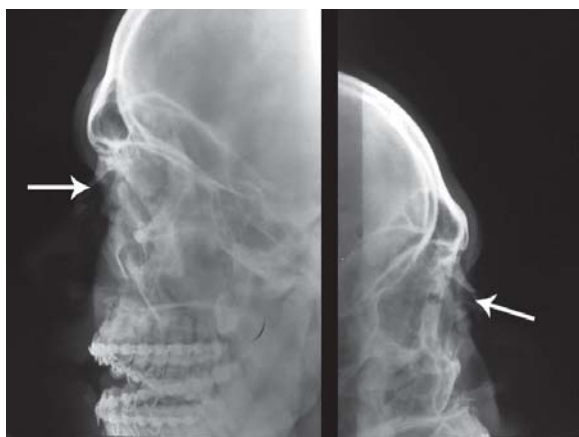


Fig. 21.9: X-ray facial skeleton lateral view showing fracture nasal bone

should be given to prevent infection (coamoxyclav + metronidazole).

- For pain relief nonsteroidal anti-inflammatory drugs are given parenterally (injection diclofenac sodium 50 mg I/M 8 hrly).
- Intraoperative and postoperative steroids (dexamethasone) may be added to reduce facial edema.
- Care of orodental hygiene by irrigation and chlorhexidine mouthwash.

Fracture of the Zygomatic Complex

Most fractures can be reduced by *Gillies temporal approach*. This method of fracture reduction is based on the anatomical fact that while the temporal fascia is attached along the zygomatic arch, the temporalis muscle runs under it and a lever inserted between fascia and muscle can slide down deep to the arch to exert its leverage (Fig. 21.10). An oblique 2 cm incision is made in temporal fossa incising deep fascia and taking care to avoid injury to superficial temporal vessels. As a pathfinder for the lever, long scissors are inserted under the fascia and slid along the surface of temporalis muscle deep to the arch. A Bristow's or Row's elevator is then inserted along the path found by the scissors so that it reaches beneath the zygomatic arch. Force is then applied in the opposite direction to the displacement of fracture and fracture is reduced. If there is associated

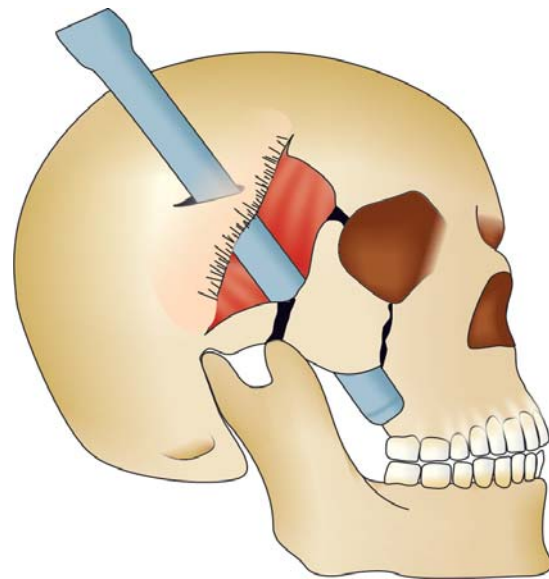


Fig. 21.10: Gillies temporal approach for reduction of zygomatic fracture

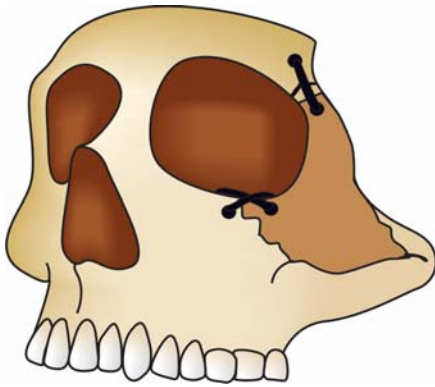


Fig. 21.11: Open reduction and internal fixation of fracture zygomatic complex

fracture of the orbital floor, then entrapment of infra-orbital soft tissues may occur during fracture reduction. It may require exploration of floor of the orbit.

If fracture of zygomatic complex is unstable, it may require open reduction and internal fixation with intraosseous wires or bone plates (Fig. 21.11).

Postoperatively, observation should be made for development of retrobulbar hematoma. The patient

presents with increasing proptosis and loss of vision requiring urgent decompression.

Fracture of Nasal Bones

These are the most commonly fractured bones of the facial skeleton. The reduction is best done within one week once swelling has settled. In case of further delay, the nasal fractures tend to fix in their displaced position. Walsham's forceps is used for disimpaction of nasal bone fracture. The blades of the forceps are closed over the nasal bone which is then mobilized with a rocking movement of the forceps first laterally and then medially to disimpact it. The external blade of the forceps should be covered with rubber tubing so as to avoid damage to the skin (Fig. 21.12). The nasal septum is then grasped with Asch's forceps and manipulated until it is straight.

Following nasal bone reduction, nasal packing is done for 2-3 days for supporting nasal bones.

A protective nasal plaster may be required for 5-7 days.

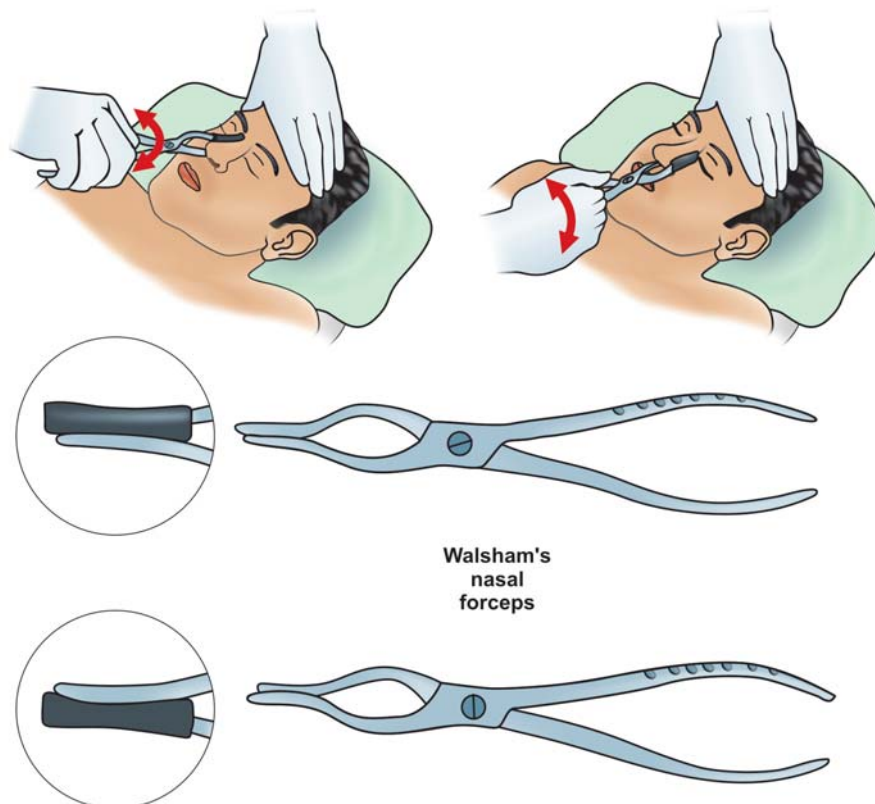


Fig. 21.12: Fracture nasal bone reduced with Walsham's forceps

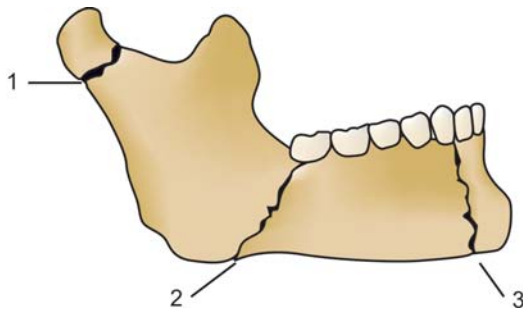


Fig. 21.13: Fracture mandible—common sites

FRACTURE OF THE MANDIBLE

Patterns of Mandible Fracture

- The common sites of fracture are: Condylar neck, angle of the mandible and body through canine sockets (Fig. 21.13).
- Fractures may occur singly or in several combinations
- Most fractures in the tooth bearing portion of the mandible are compound into the mouth because the mucoperiosteum is firmly attached to the bone and tears during injury.
- Displacement of fractured segments depend upon:
 - Direction of violence
 - Direction of muscle pull
- The muscles which elevate the mandible are all inserted *behind* the first molar, viz, masseter, medial pterygoid and temporalis.
- The muscles which depress the mandible are all inserted *in front* of the first molar, viz. geniohyoid, mylohyoid and digastric.
- Thus, most common displacement of posterior fragment is upwards and of anterior fragment downwards (Fig. 21.14).
- Another important factor deciding the displacement of angle fractures is the direction of fracture line (Fig. 21.15).
- The condylar neck is the weakest and commonest site of fracture mandible.
- The condylar head is pulled forward by the lateral pterygoid muscle leading to lateral deviation of mandible towards the side of fracture.
- If both condyles are fractured, the displacement of both heads causes the patient to gag on his molars giving an 'open bite' deformity (Fig. 21.16).

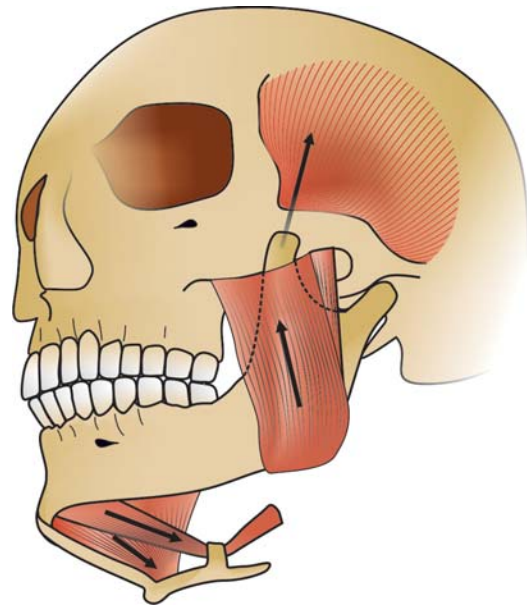


Fig. 21.14: Fracture mandible—directions of muscle pull influencing displacement of fragments

- A bilateral fracture through canine sockets detaches a midline segment from rest of the mandible (*Butterfly fracture*). This free segment will be pulled down by digastric and genioglossus muscles and tongue will fall back and occlude the airway.

TREATMENT OF MANDIBLE FRACTURE

I. Fracture of Tooth Bearing Segment

Closed Reduction with Indirect Fixation

- Fracture mandible is first *reduced* followed by *fixation*.
- Aim of *reduction* is to bring the teeth of the fractured segments into a normal relationship with those of unfractured counterpart so as to restore pre-injury dental occlusion.
- Markedly displaced fractures require general anesthesia for the fracture reduction.
- Once correct occlusion is achieved after reduction, the mandibular teeth are fixed with intermaxillary fixation (IMF).

It can be achieved by:

- a. *Eyelet wiring*: The fixing device is a stainless steel wire of 0.4 mm diameter that is doubled on itself and twisted tightly 2-3 times leaving a small loop

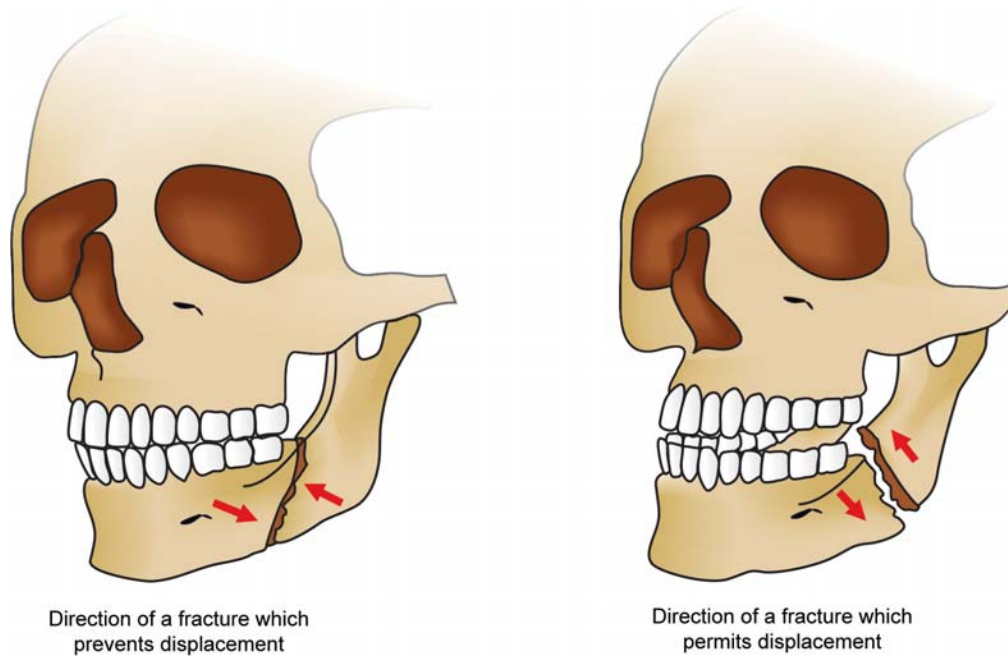


Fig. 21.15: Fracture mandible—directions of fracture lines influencing displacement of fragments

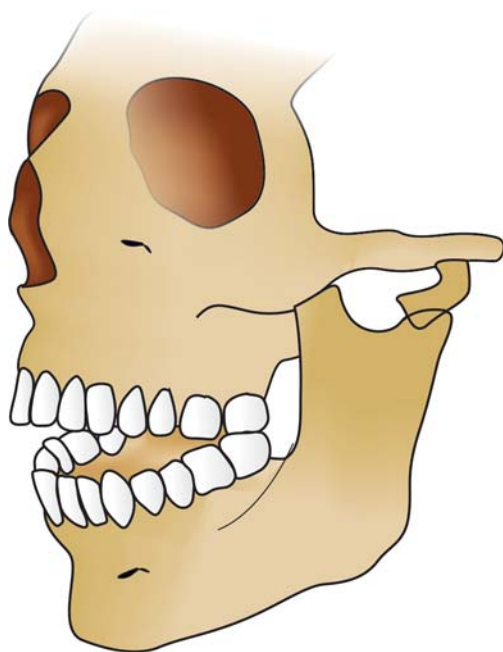
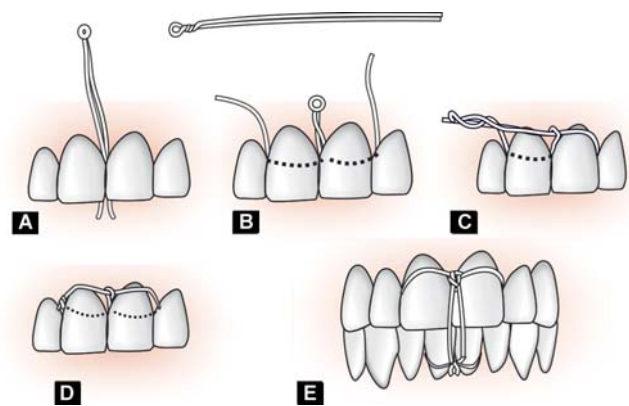


Fig. 21.16: Fracture bilateral condyles causing 'open bite' deformity

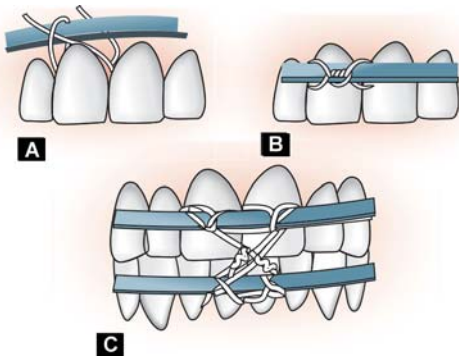


Figs 21.17A to E: Steps in eyelet wiring

wires separated and passed outwards through the next interspace and twisted together with one of the ends going through its own loop. Four or five eyelets are required for each dental arch. After eyelets have been applied to both upper and lower teeth, connecting wires are threaded through the loops to join the jaws together (Fig. 21.17).

at the end. The double wire is passed inwards between the necks of two adjacent teeth, two

b. *Arch wiring:* An arch bar (flattened soft silver bar) is moulded round the alveolar arch on its outer aspect



Figs 21.18A to C: Steps in arch wiring

at level of neck of the teeth to which it is wired. Similarly, an arch bar is applied to the maxilla and the two arch bars are wired together for effective IMF (Fig. 21.18).

- c. *Cap splinting*: In this technique cast-metal cap splints are made for the entire dentition that fit accurately over all the teeth. The splints are cemented to the teeth and in this way, provide fixation without damaging gums and teeth (Fig. 21.19).

Open Reduction with Internal Fixation

If displacement of the fracture is considerable, open reduction and internal fixation (ORIF) of the fractured segments is done with wire loop or plate. To avoid malocclusion, IMF is also done for 3 weeks (Fig. 21.20).

Various methods of ORIF are:

- a. *Transosseous wiring*

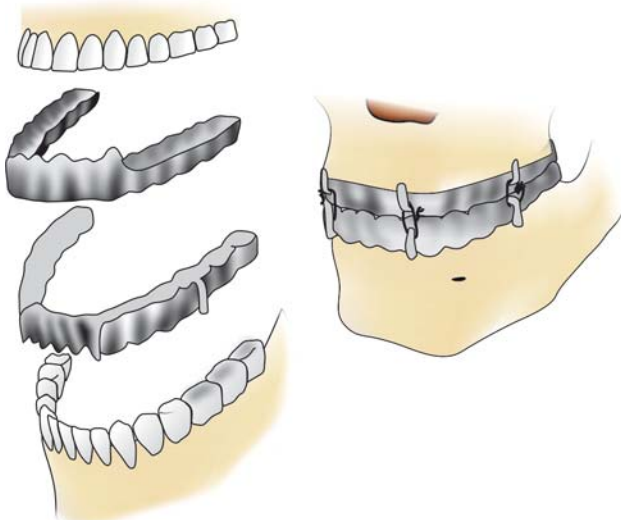


Fig. 21.19: Metal cap splints cemented to teeth

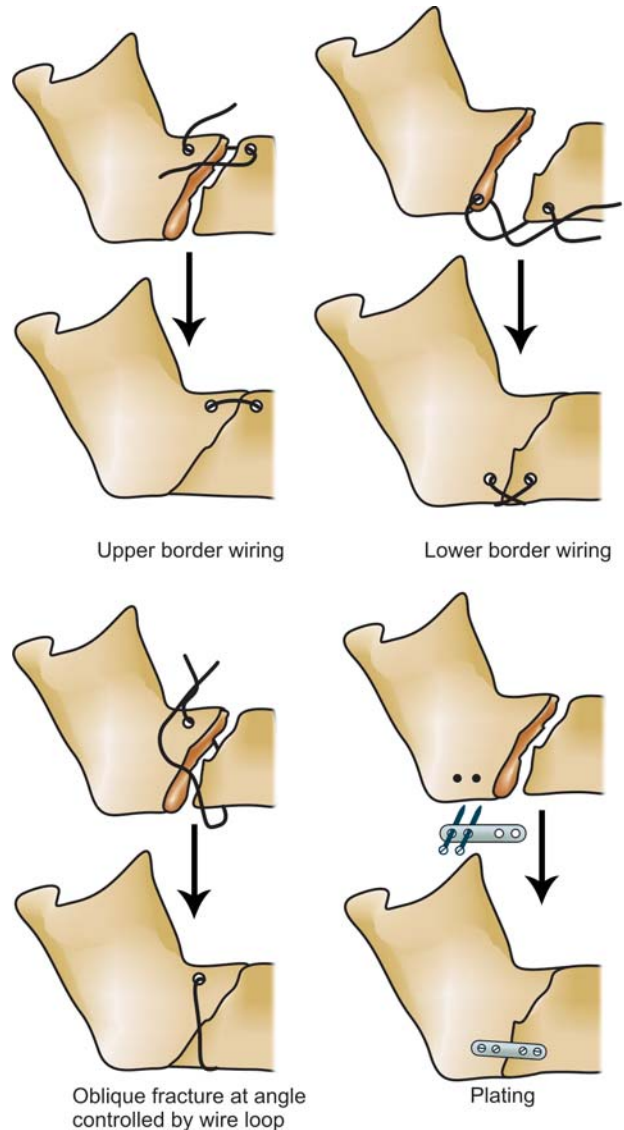


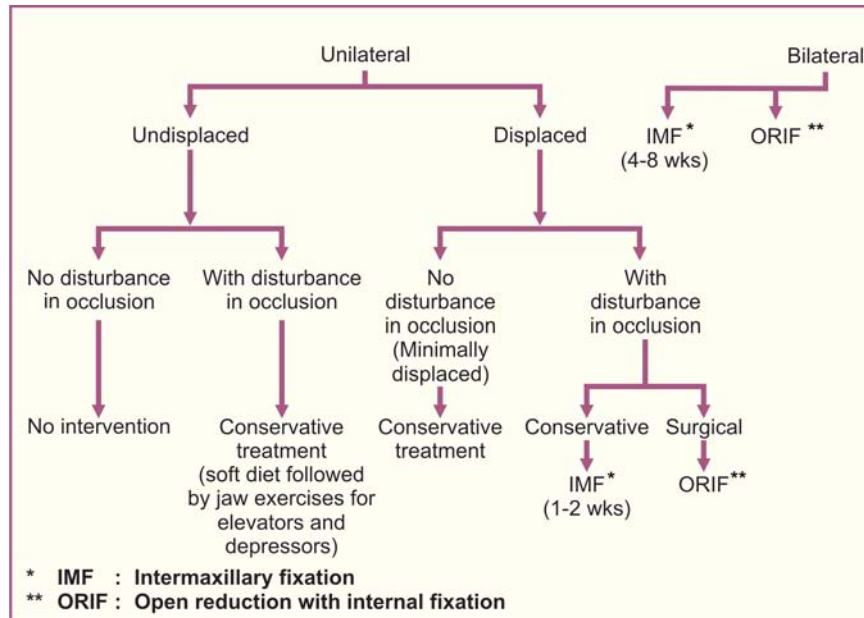
Fig. 21.20: Fracture mandible—methods of internal fixation

- b. *Mini plates*
c. *Lag screw fixation.*

II. Fracture of Non-tooth Bearing Segment

Gunning Splint

It is used for fixation of edentulous mandible. Gunning splints are like upper and lower dentures but with the teeth replaced with plastic. These splints are circumferentially wired on to the upper and lower jaws and subsequently to each other to obtain fixation.

Box 21.3: Condylar fracture management**III. Condylar Fracture**

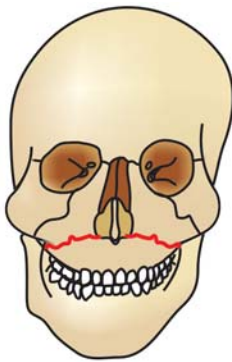
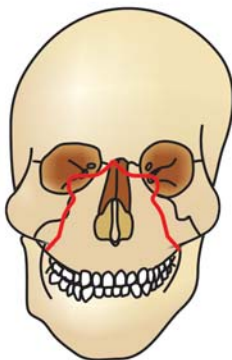
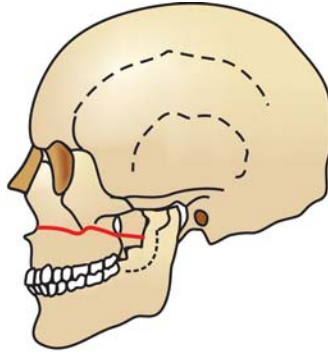
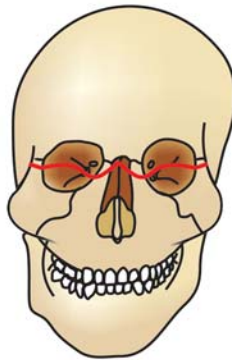
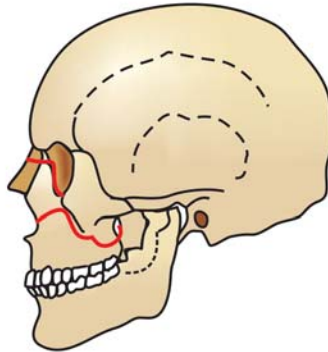
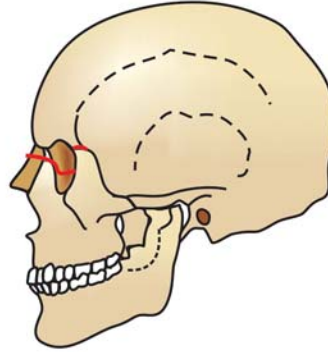
- Various treatment modalities are (Box 21.3):
 - Conservative: Soft diet, intermaxillary fixation (IMF).
 - Functional jaw exercise, e.g in condylar head fracture in children.
 - Surgical: Open reduction and internal fixation (ORIF).
- In *unilateral fracture*, there is malocclusion due to deviation of the mandible to the side of fracture. However, no attempt is made to reduce the fragment because it gets corrected spontaneously in a few days by re-education of the muscles.
- In *bilateral fracture*, there is 'open bite' deformity that is undesirable both cosmetically and functionally. Simple fixation by IMF is insufficient in such cases.
- Significantly displaced unilateral as well as bilateral fractures require open reduction and internal fixation within 7-10 days of injury.
- Direct surgical approach to condylar neck is difficult due to close proximity of parotid gland and facial nerve.
- The steps of a simple and effective surgical technique (extracorporeal technique) are:
 - Tangential incision at the angle of mandible.
 - Periosteum on both sides of ramus is raised up to as far as fracture line.
 - With a vertical cut, posterior border of mandible is removed. It gives access to the displaced condyle.
 - The condyle is removed.
 - Outside the body, two bony fragments (condyle and excised segment of mandible) are fixed with miniplates.
 - The restored bone is then returned to the patient and secured to the distal mandible with a miniplate.

FRACTURE OF THE MAXILLA**Patterns of Fracture Maxilla**

Rene Le Fort (French anatomist) classified these fractures by patterns created on cadaver skull by various degrees of force (Fig. 21.21).

Le Fort I Fracture (Horizontal Fracture)

The fracture involves the dentoalveolar component of maxilla only. Fracture line starts from anterolateral junction of pyriform aperture, passes through antero-lateral surface of maxillary antrum above canine fossa,

**Le Fort I****Le Fort II****Le Fort III****Fig. 21.21: Fracture maxilla—Le Fort classification**

moves down to zygomatic buttress and fractures the lower one third part of pterygoid plate. Thus, it separates the alveolus and palate from the facial skeleton above (Box 21.4).

Le Fort II Fracture (Pyramidal Fracture)

The fracture line passes obliquely across the maxilla on each side. Starting from the zygomatic process of

Box 21.4: Le Fort I fracture

- Swelling upper lip.
- Bleeding from nose.
- Floating maxilla (Gurien sign)
- Derangement of occlusion.
- Palatal ecchymosis.

Box 21.5: Le Fort II fracture

- Nasal deformity.
- Soft tissue swelling middle face.
- Panda face.
- Battle sign.
- Bleeding from nose
- Limitation of ocular movements.
- CSF rhinorrhea.
- Repositioning of maxilla with posterior gagging.

maxilla, the fracture line goes upwards and medially to the infraorbital margin and then across the root of the nose to meet a similar fracture line from the opposite side. The orbital floor is always involved. Posteriorly, the fracture line continues through the lateral wall of maxillary antrum at a higher level than Le Fort I to the pterygoid plates at the back (Box 21.5).

Le Fort III Fracture (Craniofacial Dysjunction)

The fracture line passes high up through back of the nose in ethmoid area, back of both orbits and through both zygomatic arches. Posteriorly, the nasal septum is fractured high up and likewise the pterygoid processes. There may be CSF rhinorrhea due to involvement of cribriform plate. There is separation of facial skeleton from the base of the skull (Box 21.6).

Treatment of Fracture Maxilla

- Associated head injury, cervical spine injury or other serious injury should be given priority and treated first.

Box 21.6: Le Fort III fracture

- Dish faces appearance.
- Hooding of eyes.
- Enophthalmos.
- CSF rhinorrhea.
- Panda face.
- Battle sign.
- En bloc mobility of whole facial skeleton.



Fig. 21.22: Closed reduction of fracture maxilla using Rowe's disimpaction forceps

- However, fractures of middle third of face should be treated with minimum delay as they tend to fix rapidly in their displaced position.
- The aim of treatment is fracture reduction (to restore normal occlusion), fixation and immobilization.
- In fresh fractures of Le Fort I type, closed reduction by manipulation can be done. It is done with Rowe's disimpaction forceps that grasps the palate between the nasal and palatal mucosa. Sometimes considerable force is required in downward, forward and sideways movements to disimpact the maxilla (Fig. 21.22). It is followed by intermaxillary fixation for achieving occlusion with the mandible.
- In delayed cases, open reduction and internal fixation of the fracture is done.
- *Bicoronal flap incision:* It is used for stabilization of upper part of the face. The incision starts from the front of one ear, goes across the vault of skull (high in the hair line) and then to the front of other ear. The flap is reflected down till supraorbital ridges are exposed. This incision exposes nasal bones, lateral orbital rim, frontal bones and zygomatic arches. All the fractured bones are reduced and fixed with stainless steel wires or titanium microplates. The bone deficiencies can be filled with bone grafts or titanium mesh.
- *Blepharoplasty or transconjunctival incision:* It is used for stabilization of midface. The incision is made in

the lower eyelid or lower conjunctival sac. It exposes fractures of the infraorbital rim or orbital floor. The fractures are reduced and fixed in the same way as described above.

- *Vestibular incision:* It is used for stabilization of lower part of maxilla. The incision is made in the gingival sulcus above the maxillary teeth as far back as the second molar tooth. The fracture is reduced and fixed with plates and wires. The dental arch is restored to its original shape and IMF is done using eyelet wires or dental arch bars to achieve normal occlusion.
- With the availability of maxillofacial plating system, external fixation with pins, POP headcaps and haloframes are rarely used these days. However, external fixation is still indicated in case of multiple and unstable fragments of maxilla. The mandible is fixed to the cranium with the maxilla as a 'sandwich' between the two. Pins are used for cranial fixation and mandibular fixation. Then all the cranial pins are connected to mandibular pins with connecting bars.

ORBITAL BLOW OUT FRACTURE

Blunt trauma, e.g. elbow hitting the eyeball can push it back within the orbit. It leads to increase in intraorbital pressure that fractures the floor of the orbit into the maxillary antrum without involving the orbital rim. Thus, orbital contents (fat and muscles) herniate down into the maxillary antrum causing enophthalmos. In case inferior oblique and inferior rectus muscles are caught in the fracture, it causes diplopia due to restriction in the movements of eyeball.

X-ray of maxillary sinus shows 'hanging drop appearance'. Such fractures should be treated within 10 days of injury. The floor of orbit is approached through blepharoplasty incision. The periorbital soft tissues are gently separated from the fractured bone and the defect in orbital floor is made up with bone graft or titanium mesh. Sometimes packing of the maxillary antrum via Caldwell-Luc approach is required if the fragments are unstable.

COMPLICATIONS OF MAXILLOFACIAL FRACTURES

- Infection of maxillary sinus.
- Osteomyelitis.

- Meningitis due to CSF leak.
- Cavernous sinus thrombosis.
- Malocclusion of teeth.
- Ankylosis of TM joint.
- Anesthesia and paresthesia
 - ☐ In lower lip (inferior dental nerve injury).
 - ☐ In upper lip, side of nose, lower eyelid (infra-orbital nerve injury).
- Facial nerve injury.
- *Superior orbital fissure syndrome*: In malunited zygomatic complex fractures, there is damage to the contents of superior orbital fissure. Third, fourth and fifth cranial nerves are affected leading to ophthalmoplegia, proptosis and retrobulbar pain.
- Malunion, nonunion and delayed union.
- Nasal blockage due to deviated nasal septum.
- Epiphora due to damage to nasolacrimal duct.
- Anosmia due to olfactory nerve damage.

22

Chapter

Cleft Lip and Cleft Palate

Sanjay Marwah

EMBRYOLOGY OF LIP AND PALATE

Face develops from the cranioneural crest cells. At 5th week of intrauterine life, frontonasal process divides into right and left parts. Each of the two parts again divides into two processes:

- Lateral nasal process
- Medial nasal process.

The two medial nasal processes join to form the *median process* or *Processus globularis* (Fig. 22.1). The processus globularis meets and fuses with maxillary process to form the upper lip. So, processus globularis forms the central part of nose and central part of upper lip. Lateral nasal process forms the ala of nose. The maxillary process forms the lateral part of upper lip and mandibular process forms the lower lip (Fig. 22.2).

The palatal shelves form as medial outgrowths from the maxillary processes. These grow medially and fuse with each other to form the palate.

ANATOMY OF LIP AND PALATE

- The junction of upper lip with skin is known as white line (Fig. 22.3).
- Below the white line, the dark part of lip is known as red line or vermillion.
- The central part of white line is known as Cupid's bow.
- The prominent point on middle of white line is known as median philtral tubercle.
- Above white line, two vertical columns on paramedian areas are known as philtral columns.
- The pentagonal area thus formed is called philtrum.
- The nasal opening has central columella and two openings on either sides (nostrils).

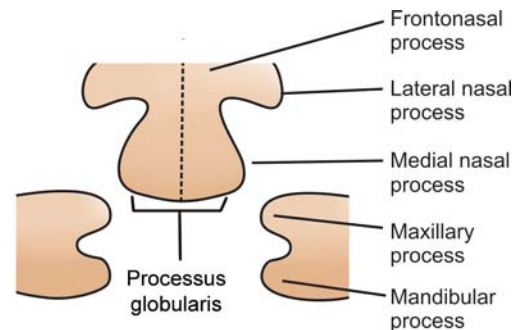


Fig. 22.1: The head of human embryo at five weeks

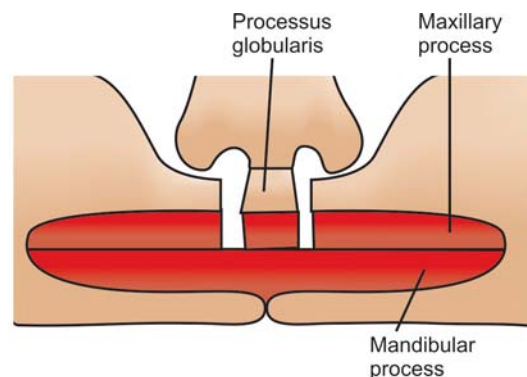


Fig. 22.2: Formation of face by fusion of various processes

- The premaxilla (anterior maxilla and four incisors), together with the hard palate anterior to incisor foramen, is termed as the primary palate.
- The remaining hard palate and soft palate are known as secondary palate.

CLEFT LIP

- It occurs as a result of defect in fusion of processus globularis with maxillary process.
- Thus in upper lip, the cleft is always on one side of midline and not in the midline (Fig. 22.4).

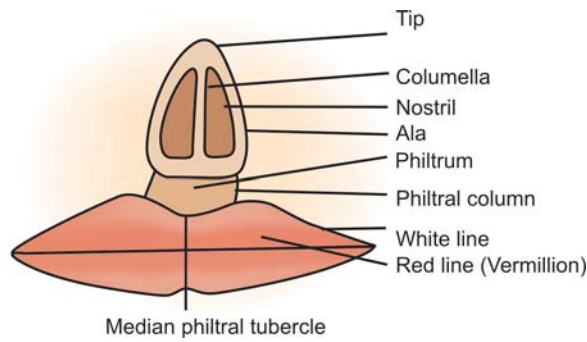


Fig. 22.3: Anatomy of the lip



Fig. 22.4: Cleft lip left side

- In lower lip, if mandibular processes fail to fuse in midline, it produces median cleft lip. It occurs very rarely.

CLEFT PALATE

It occurs due to failure of fusion of palatal processes of maxilla and occurs in midline (Fig. 22.5).

Incidence

- Isolated cleft lip is three times more common than isolated cleft palate.
- Isolated cleft lip is more common in males.
- Isolated cleft palate is more common in females.
- Cleft is unilateral in majority of cases.
- In unilateral cleft lip, cleft is on left side in majority of cases.



Fig. 22.5: Cleft palate

Etiology

- Environmental factors:*
 - Vitamin B₆ deficiency
 - Radiation exposure
 - Drugs (steroids, insulin)
- Genetic factors:* If parents are affected with cleft, risk of first child having cleft is 4% and in second child, risk increases to 17%.

Associated Anomalies

There can be anomalies involving heart, skull, nervous system, extremities. In **Pierre-Robin syndrome**, there is associated short mandible.

Classification

- The cleft may involve lip, palate or both (Fig. 22.6).
- The cleft lip may be unilateral or bilateral.
- The cleft may be complete or incomplete.
- Any classification should be simple and should be able to describe site, size, extent and type of cleft.
- **LAHSHAL system** is an example of such classification.
 - The capital words L, A, H and S represent complete cleft of lip, alveolus, hard and soft palate respectively.
 - Thus LAHSHAL represents complete bilateral cleft lip and palate (Fig. 22.7).
- Another simple way of classification is **Balakrishnan grouping**:
 - Group-I Cleft lip only
 - Group-II Cleft palate only
 - Group-III Cleft lip and palate together



Fig. 22.6: Bilateral cleft lip and palate

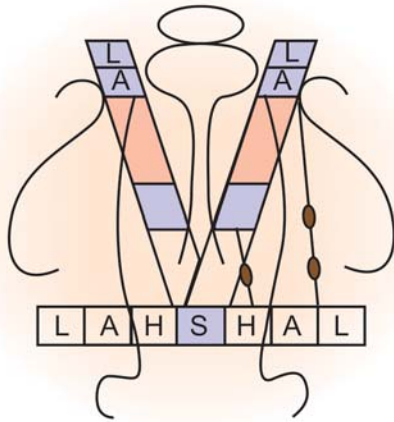


Fig. 22.7: LAHSAL system of anatomical classification

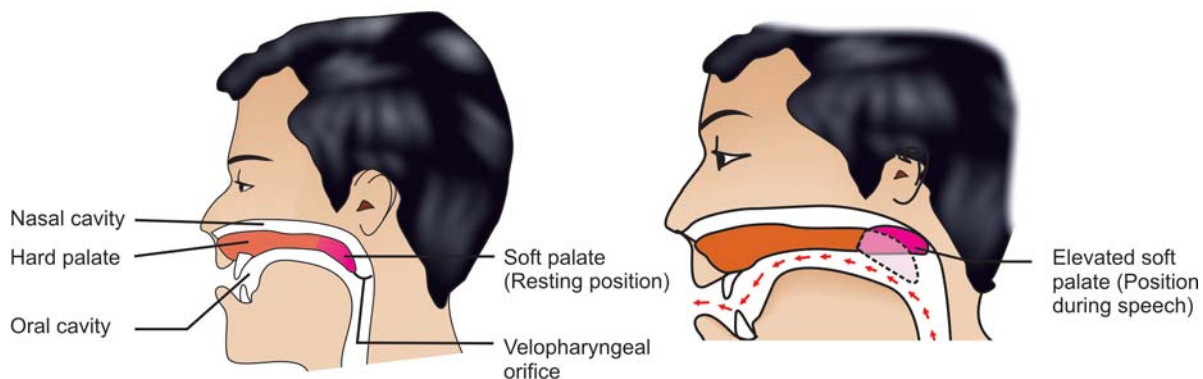


Fig. 22.8: Position of soft palate during rest and during speech

Problems

Cleft Lip

- Cosmetic problem.
- Psychological problem secondary to cosmetic appearance.

Cleft Palate

The problems are primarily functional. The palate provides a partition between oral and nasal cavity. During speech and swallowing, the soft palate elevates and forms an airtight seal with lateral and posterior pharyngeal wall (Fig. 22.8). It prevents airflow into nasopharynx and gives normal speech. Elevation of soft palate also prevents regurgitation of food and drinks into the nasopharynx. Thus, cleft palate will cause velopharyngeal incompetence leading to:

- Defective speech (nasal twang).
- Nasal regurgitation of food.

Other problems associated with cleft palate are:

- Abnormal facial growth.
- Abnormal dentition.
- *Hearing problem:* It is because soft palate muscles help in opening and closure of eustachian tube to equalize pressure within the middle ear. Impairment of this mechanism leads to accumulation of fluid in the middle ear (glue ear) that can get infected leading to otitis media.

Aims of Surgical Repair

Cleft Lip

To give cosmetically acceptable face so that there is no psychological problem for parents or the patient.

Cleft Palate

- To provide intact roof to the mouth and a mobile soft palate so that it reaches posterior pharyngeal wall on phonation and produces competent nasopharyngeal sphincter.
- To give well-aligned teeth and no loss of hearing.

Time of Surgical Repair

Cleft Lip

Repair is done as early as possible to take care of cosmetic problem. It is done at 3 months of age (Rule of Ten, is shown in Box 22.1).

Box 22.1: Rule of 10

10 weeks age
10 pounds weight
10 gm% hemoglobin
10,000/cmm TLC

Cleft Palate

The repair is delayed for some time since it is a functional problem. It is done at 1½ years of age when the child learns to speak. Moreover, if repair is done early, there is risk of retarded maxillary growth due to surgical trauma. The problem of nasal regurgitation is tackled by spoon feeding or by bottle feeding that has long nipple with a big hole.

Surgical Repair of Cleft Lip

In cleft lip, all abnormalities are on the cleft side that has medial and lateral portions. Basic problem is deficiency of tissues on medial side of cleft and extra tissue on lateral side of cleft (Fig. 22.9). So, principle of repair is to bring extra tissue from lateral side to the medial side so as to produce bilaterally symmetrical upper lip.

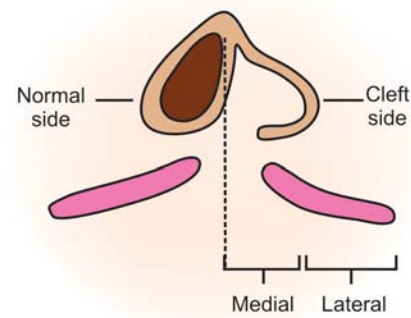


Fig. 22.9: Deficient lip tissues in medial part of cleft side

Steps of Repair

- Repair is done under general anesthesia.
- 0.5% xylocaine with 1 : 100,000 adrenaline is infiltrated locally for
 - Keeping general anesthesia at lighter plane.
 - Making tissues more prominent.
 - Hemostasis.
- Skin markings are made and paring of edges of the cleft is done.
- Accurate repair of skin, muscles and mucous membrane is done.
- The continuity of white line should be maintained and 'Cupid's bow' should be preserved.
- Aim of repair is to give a long zigzag scar that does not contract to produce notching.
- The closure can be done using various techniques:
 - *Millard technique*: It is rotation advancement type of repair. The medial flap is rotated down and lateral flap is advanced to fill the gap (Fig. 22.10).
 - *Tennison technique*: It is a form of triangular flap repair. A triangular flap is brought from lateral side of cleft to medial side.

Surgical Repair of Cleft Palate

- Repair is done under general anesthesia.
- Local infiltration of xylocaine with adrenaline.
- Paring of the edges of the cleft.
- Raising of mucoperiosteal flaps on each side of the cleft.
- Relaxation incisions on lateral sides to help medial movement of these flaps so that they meet in the midline without tension.

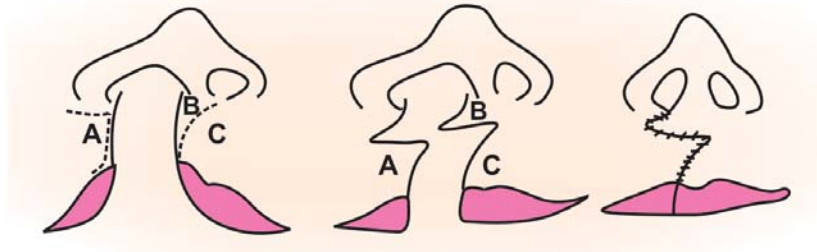


Fig. 22.10: Cleft lip repair—Millard's technique

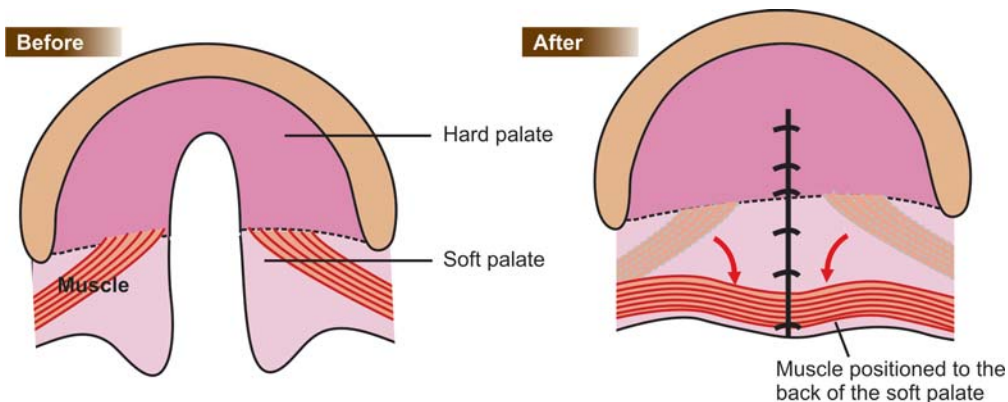


Fig. 22.11: Cleft palate repair

- Soft palate muscles are dissected from bony edge of the posterior hard palate, realigned transversely and sutured.
- The nasal mucosal lining is sutured.
- The oral mucosal lining is sutured (Fig. 22.11).

Complications of Surgical Repair

- Respiratory obstruction—mostly seen in case of micrognathia and may require tracheostomy.
- Hemorrhage from posterior palatine artery.
- Infection.
- Breakdown of suture line.
- Asymmetrical Cupid's bow.

The overall management of cleft lip and cleft palate requires a team approach. The aim of the surgery is that the child should “look well, eat well and speak well”. In cleft palate patients, speech therapy is required after surgery. Hearing problems require ENT specialist and dental problems of occlusion require care by orthodontic surgeon. In many cases, secondary operations are

required during later life to improve appearance and functions.

The outline of management of cleft lip and cleft palate is given in Box 22.2.

Box 22.2: Outline of management of cleft lip and cleft palate

- Repair of cleft lip—at 3 months
- Repair of cleft palate—at 1½ years.
- Pharyngoplasty for speech correction (if required)—at 3 years.
- Orthodontic preparation for bone grafting (in cleft of alveolus)—at 8-9 years.
- Bone grafting of alveolus—10 years.
- Definitive orthodontics—12-14 years.
- Maxillary osteotomy (for maxillary retrusion)—17-18 years.
- Rhinoplasty (for nasal deformity)—17-18 years.
- Lip revision (if necessary)—17-18 years.

23

Chapter

The Thyroid Gland

Sham Singla, Sanjay Marwah

SURGICAL ANATOMY

The thyroid gland develops from a median bud that descends from an opening at base of the tongue (foramen caecum) as thyroglossal duct. The lower end of the duct grows and divided into two lobes. The thyroglossal duct disappears and a remnant remains as a pyramidal lobe.

The thyroid gland weighs about 25 grams. It is situated in lower part of the front of the neck. It has right and left lobes connected by isthmus in the midline. It lies against C₅-T₁ vertebrae claspings the upper part of trachea. It has a true capsule which is condensation of peripheral connective tissue of the gland and a false capsule derived from pretracheal layer of deep cervical fascia (Fig. 23.1). The latter is thickened on inner surface of the gland forming a suspensory ligament (**Ligament of Berry**) on each side, which is attached above to the cricoid cartilage. The thyroid gland moves on deglutition due to this ligamentous attachment.

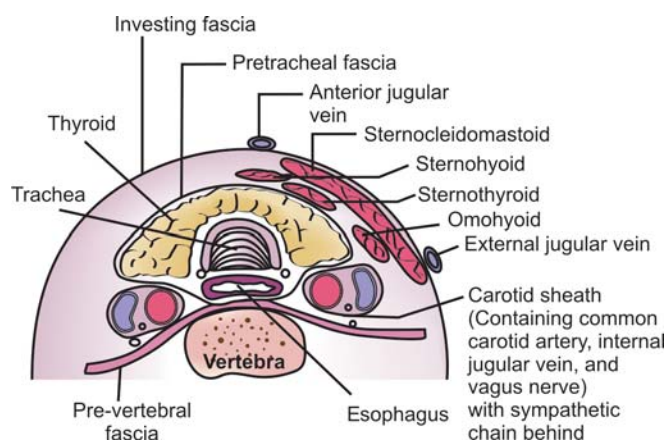


Fig. 23.1: Transverse section of neck (C 6 level) showing relations of thyroid gland

Arterial supply is mainly by superior (branch of external carotid artery) and inferior (branch of thyrocervical trunk) thyroid arteries (Fig. 23.2). There may be additional supply from thyroidea ima artery (from brachiocephalic trunk or arch of aorta).

Venous drainage is by superior (drains in internal jugular vein), middle (drains in internal jugular vein) and inferior (drains in innominate vein) thyroid veins (Fig. 23.2).

The **lymphatics** follow the arteries and drain mostly into anterosuperior and posteroinferior groups of the deep cervical nodes and also to pretracheal nodes (Delphic nodes).

The **nerves** in relation to thyroid gland are superior laryngeal nerve and recurrent laryngeal nerve (both branches of vagus nerve). The superior laryngeal nerve descends along superior thyroid vessels at upper pole and divides into external and internal laryngeal nerves. The internal laryngeal nerve is sensory to the larynx above vocal cords and external laryngeal nerve supplies the

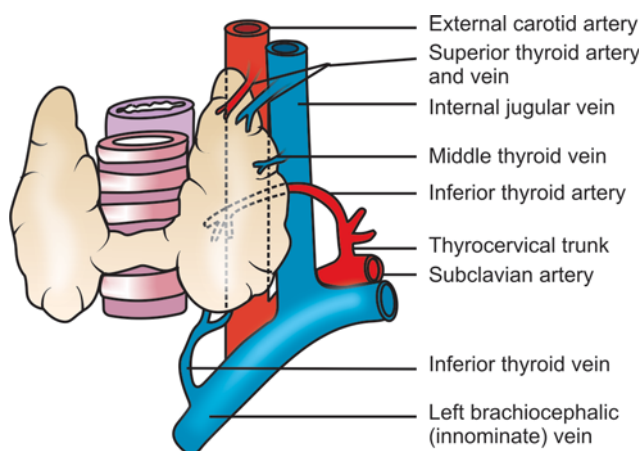


Fig. 23.2: Blood supply of thyroid gland

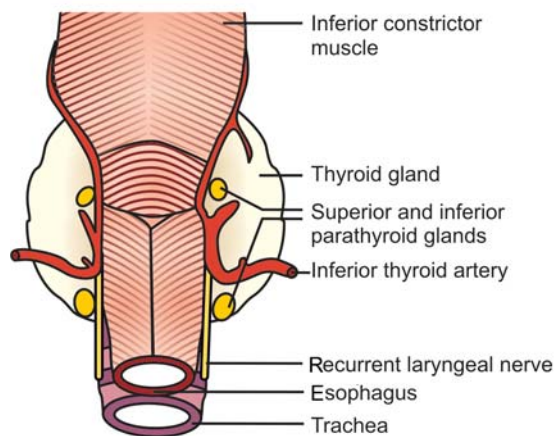


Fig. 23.3: Posterior view of thyroid gland showing recurrent laryngeal nerve

cricothyroid muscle. The recurrent laryngeal nerve is sensory below vocal cords and motor to all laryngeal muscles except cricothyroid. On right side it loops the subclavian artery and on left side, the arch of aorta. It then ascends in tracheo-esophageal groove and enters larynx (Fig. 23.3). In 1% cases it may be non-recurrent on right side and runs horizontally coming behind carotid sheath.

The relation of nerves and vessels to the thyroid gland at its poles is like inverted-V (Λ) (Fig. 23.4). So, superior thyroid vessels should be ligated as near the upper pole as possible and inferior thyroid artery should be ligated as far from lower pole as possible to avoid damage to adjoining nerves. Injury to external laryngeal nerve leads to loss of pitch of the voice while injury to recurrent laryngeal nerve leads to hoarseness of voice.

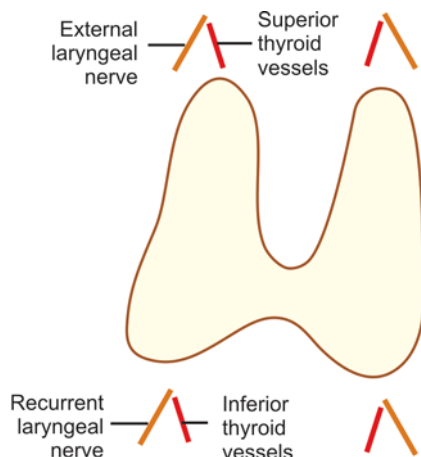


Fig. 23.4: The relation of nerves and vessels at upper and lower poles of thyroid

PHYSIOLOGY

The thyroid gland has two types of secretory cells: follicular and parafollicular cells. The follicular cells secrete the thyroid hormones tri-iodothyronine (T_3) and tetraiodothyronine (T_4); the parafollicular or C-cells secrete thyrocalcitonin.

The steps of synthesis of thyroid hormones are as follows:

- I. **Trapping** of inorganic iodide from blood.
- II. **Oxidation** of iodide to iodine.
- III. Iodine **binding** with tyrosine to form iodotyrosine.
- IV. **Coupling** of one monoiodotyrosine (MIT) and one diiodotyrosine (DIT) to form T_3 or two DIT join to form T_4 . T_3 and T_4 remain attached to thyroglobulin in the thyroid gland. On requirement, T_3 and T_4 are released in blood and get bound to serum proteins. A small amount of hormone remains free in the serum and is biologically active.

Antithyroid drugs act by blocking various steps of synthesis of thyroid hormones (Box 23.1).

Box 23.1: Antithyroid drugs blocking synthesis of thyroid hormones

Steps of synthesis	Blocking drugs
Trapping	Thiocyanates, Perchlorates
Oxidation	Carbimazole, Propyl thiouracil
Binding	Carbimazole
Coupling	Carbimazole

HYPOTHALAMIC—PITUITARY THYROID AXIS

Release of TRH from hypothalamus stimulates anterior pituitary to release TSH, which in turn stimulates thyroid to release T_3 and T_4 . The rising levels of T_3 and T_4 have negative feedback effect on anterior pituitary as well as hypothalamus (Fig. 23.5).

THYROID FUNCTION TESTS

Most of the thyroid function tests performed in the past are only of historical importance. As a routine, only a small number of tests need to be performed.

Serum TSH, T_3 and T_4 Levels

If TSH level is within normal range, it is suggestive of euthyroid state and estimation of T_3 and T_4 is not required.

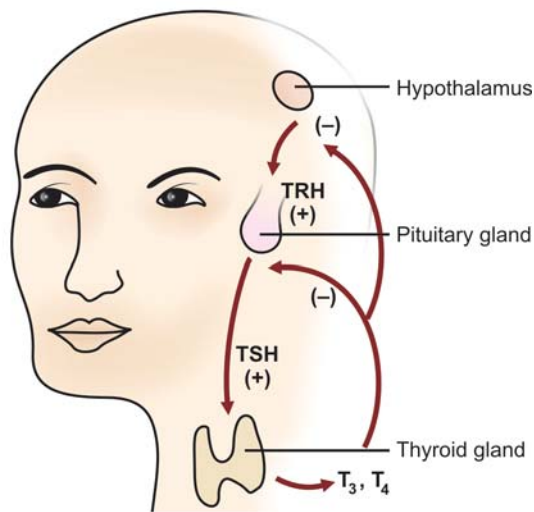


Fig. 23.5: Hypothalamic—pituitary thyroid axis

In euthyroid patient, T_3 , T_4 and TSH levels will be within normal range. In hyperthyroid state, T_3 and T_4 levels will be high and TSH levels are on lower side (even undetectable) due to increased negative feedback on pituitary gland.

In hypothyroid state, T_3 and T_4 levels will be low and TSH levels will rise due to decreased negative feedback on pituitary gland (Box 23.2).

Normal values:	T_3	–	3.5-7.5 p mol/lit.
	T_4	–	10-30 n mol/lit.
	TSH	–	0.3-3.3 mμ/lit.

Box 23.2: Hormone levels

	T_3	T_4	TSH
Euthyroid	N	N	N
Hypothyroid	↓	↓	↑
Hyperthyroid	↑	↑	↓

Isotope Scan

I^{123} or Tc^{99m} is given in low dosage and its pick up by active thyroid tissue is demonstrated with gamma camera. The findings in a case of thyroid nodule can be as follows:

- Warm nodule:** Nodule as well as surrounding normal thyroid tissue has normal and equal uptake of the isotope.
- Cold nodule:** Nodule has no uptake while surrounding thyroid tissue has normal uptake of the isotope (Box 23.3).

Box 23.3: Cold nodule: Causes

- Cysts.
- Fibrosis.
- Hemorrhage.
- Carcinoma.

- Hot nodule:** Nodule has increased uptake while surrounding thyroid tissue has decreased uptake of isotope. It is suggestive of autonomous toxic nodule.

The malignant nodules are mostly cold, but can also be hot sometimes. Therefore, isotope scan is not diagnostic of thyroid malignancy.

- In toxic MNG, isotope scan demonstrates whether nodules or inter-nodular area are hyperactive that helps in deciding the therapy.
- Isotope scan is useful in locating ectopic thyroid tissue (lingual, subhyoid, mediastinal, ovarian) and to look for retrosternal extension of thyroid gland.
- Whole body isotope scanning is useful in locating metastases of thyroid malignancy since these areas have functional activity. However, normal thyroid gland has to be removed (with surgery or radio-iodine ablation) before the scan is done since functional thyroid metastasis cannot compete with normal thyroid tissue in iodine uptake.

Thyroid Antibodies

Antibodies against thyroid are useful in determining the cause of thyroid dysfunction. The levels are high in autoimmune thyroiditis and Graves' disease.

HYPOTHYROIDISM

It is a clinical syndrome that results from deficiency of or resistance to, thyroid hormones.

In children, it results in decreased growth and mental retardation.

In adults, it leads to generalized slowing of body processes and development of myxoedema (see Fig. 23.25).

Causes

1. Primary

a. Thyroiditis

- Autoimmune thyroiditis
- Subacute thyroiditis
- Hashimoto's thyroiditis
- Riedel's thyroiditis
- Primary myxoedema

- b. *Iatrogenic*
 - Radioiodine
 - Surgery
 - Antithyroid drugs.
 - c. *Endemic*
 - Iodine deficiency
 - d. *Congenital*
 - Thyroid dysgenesis
 - Dyshormonogenesis
 - e. *Goitrogens*
2. **Secondary**
- a. Hypopituitarism
 - b. Hypothalamic hypothyroidism
3. **Peripheral resistance to thyroid hormones**

Symptoms

Symptoms are nonspecific and insidious in onset. These are tiredness, weight gain, cold intolerance, constipation and menstrual irregularities.

Signs (starting from head to toe)

- Dry, coarse hair
- Periorbital puffiness
- Hoarse voice
- Bradycardia
- Cold extremities
- Cardiomegaly
- Pericardial effusion
- Delayed relaxation of ankle jerk (diagnostic)

Investigations

- Low T₃ and T₄ with high TSH levels.
- In pituitary disease, TSH levels fail to rise in response to TRH stimulation.
- High levels of thyroid antibodies in autoimmune thyroiditis.

Treatment

It is simple and effective. Patients usually require lifelong thyroid hormone replacement.

Oral thyroxine (0.1-0.2 mg) is given once a day. In elderly patients with cardiac disease, start with 0.05 mg of thyroxine per day. Improvement is seen in 1-2 weeks and most signs and symptoms disappear in few months. Adequacy of response is assessed by clinical signs and biochemical assay.

Myxoedema

Myxoedema is the term applied for severe thyroid failure and the patient has accentuated signs and symptoms of hypothyroidism. Myxoedema appearance (puffy face, pouting lips, malar flush) is due to accumulation of mucinous edema. Dry skin and yellow hue is due to decreased conversion of carotene to vitamin A. In neglected cases, patient may develop hypothermia, hypotension, bradycardia and even myxoedema coma can occur which carries a high mortality. The precipitating factors are infection, surgery, drugs (sedatives) and hypothermia. The treatment includes slow rewarming, intravenous T₃ and hydrocortisone.

GOITER

The generalized enlargement of thyroid gland is goiter. The term goiter is derived from latin word “guttur” that means “the throat”. WHO goiter grading system is as follows:

- Grade 0 - No palpable/visible goiter
- Grade 1 - Palpable goiter/visible on neck extension
- Grade 2 - Goiter visible in normal neck position
- Grade 3 - Very large goiter

Morphologically, it can be:

- Diffuse enlargement
- Multinodular goiter (MNG)
- Solitary thyroid nodule (STN)

Functionally, it can be:

- Euthyroid
- Hyperthyroid
- Hypothyroid

Classification of goiter is given in Box 23.4.

Box 23.4: Classification of goiter

Simple goiter

- Diffuse hyperplastic
- Colloid
- Multinodular

Toxic goiter

- Diffuse (Graves' disease)
- Multinodular (Plummer's disease)
- Toxic adenoma

Neoplastic

- Benign
- Malignant

Inflammatory

- Autoimmune (Hashimoto's thyroiditis)
- Granulomatous (de Quervain's thyroiditis)
- Fibrosing (Riedel's thyroiditis)
- Infective (bacterial, viral)

Box 23.5: Prophylaxis against goiter

- | | | |
|---|---|---------------------|
| • Endemic goiter | — | Iodized salt |
| • Puberty goiter | — | Thyroxin supplement |
| • Avoid goitrogens (Brassica, cabbage, drugs) | | |

Prophylaxis against goiter is given in Box 23.5.

Stages in Goiter Formation

1. Whenever there is rise in TSH, there is increased stimulation of thyroid gland leading to diffuse thyroid hyperplasia. All the thyroid lobules are active.
2. At a later stage, many lobules become inactive and full of colloid (stage of colloid goiter).
3. Later on due to fluctuation in TSH levels, areas of active and inactive lobules develop in thyroid gland.
4. Active lobules may undergo hemorrhage (due to increased vascularity) that follows necrosis and fibrosis leading to formation of nodules. These nodules are inactive while internodular area has active lobules.

Diffuse Hyperplastic Goiter

It is usually seen at times of increased physiological demands (e.g. puberty, pregnancy, lactation) that cause increased TSH stimulation. The thyroid gland is diffusely enlarged in shape of a butterfly and is soft in consistency. If TSH stimulation ceases, the goiter may regress. If TSH stimulation persists, diffuse hyperplastic goiter changes to **colloid goiter** where all acini are distended with colloid (Fig. 23.6). The thyroid swelling characteristically moves on deglutition (Fig. 23.7).



Fig. 23.6: Colloid goiter in lower neck



Fig. 23.7: Colloid goiter moving up with deglutition



Fig. 23.8: Multinodular goiter

In endemic areas, incidence of goiter can be significantly reduced by supplementing iodized salt in the diet. In early stages, 0.1-0.2 mg of daily thyroxin may cause regression of the hyperplastic goiter in a few months time.

Multinodular Goiter (MNG)

It is the end stage of hyperplastic goiter and is irreversible. It is more common in females and usually presents in 4-5th decade of life.

Symptoms and Signs

- Asymptomatic neck mass (Fig. 23.8).
- Dyspnea and dysphagia may occur in a large MNG due to compression of trachea and esophagus respectively.
- Firm nodular thyroid that moves on deglutition.

Complications

- A rapidly appearing painful nodule is usually due to **hemorrhage**.
- Area of hardness and irregularity may occur which could be due to **calcification** or **malignant change**.
- 4-10% of MNG may undergo malignant change and it is usually follicular carcinoma.
- Features suggestive of malignant change in MNG are:
 - ☐ Rapid painless enlargement (Fig. 23.9)
 - ☐ New solitary nodule
 - ☐ Fixation and hardness of goiter
 - ☐ Hoarseness of voice
 - ☐ Appearance of neck nodes
- 10-20% cases of MNG may have **secondary thyrotoxicosis**.

Progress and outcome of goiter is shown in Box 23.6.



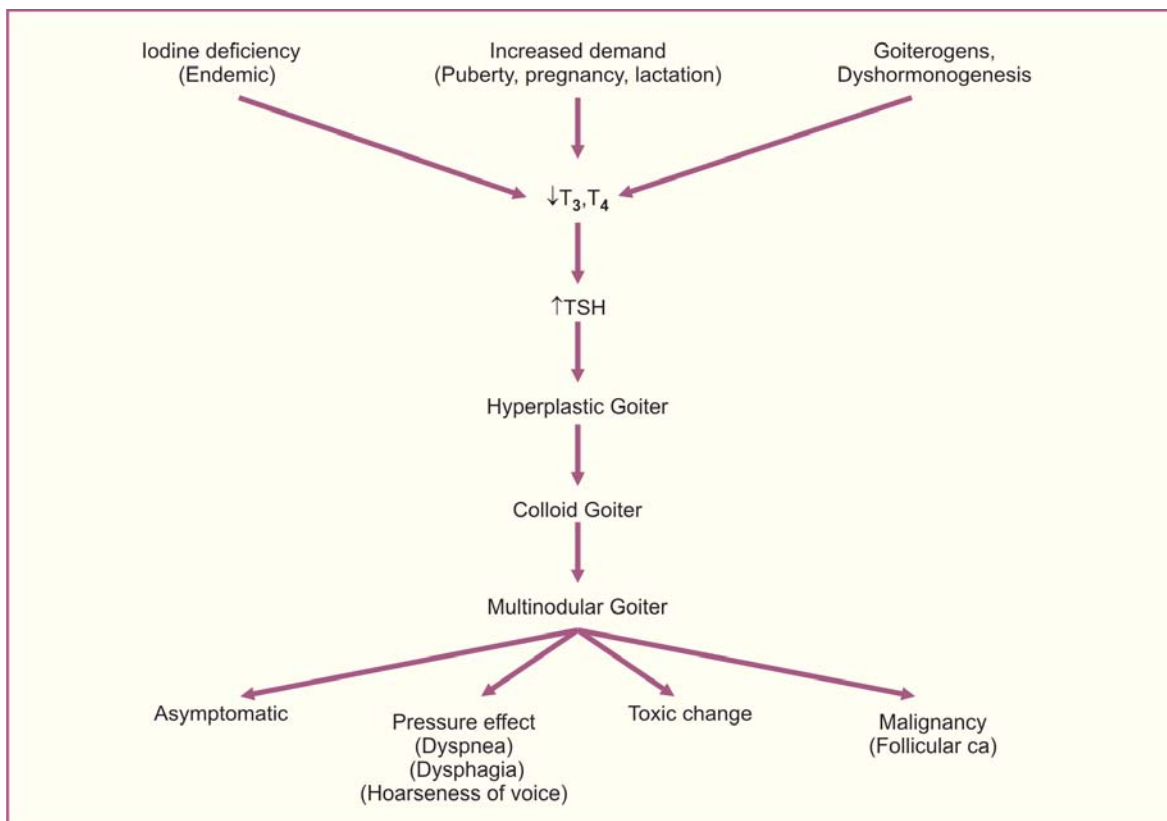
Fig. 23.9: Rapid painless enlargement in multinodular goiter

Investigations

- Routine investigations (Hb, BT, CT, Urine)

- Blood urea, blood sugar
- ECG, chest X-ray
- *X-ray soft tissue neck:* AP and lateral view are done to look for tracheal deviation and tracheal compression (scabbard trachea*) respectively. It is

Box 23.6: Progress and outcome of goiter



*Scabbard literally means sheath of a sword that is compressed anteroposteriorly.

important from anesthetist's point of view while doing endotracheal intubation during anesthesia.

- *Indirect laryngoscopy*: To see and document the status of vocal cords (for medicolegal reasons).
- T_3T_4 and TSH levels.
- FNAC of thyroid nodule to rule out malignancy.

Treatment

Surgery is the mainstay of treatment.

Indications for surgery are:

- Cosmetic reasons
- Pressure symptoms
- Risk of malignant change

Types of Surgery

1. *Subtotal thyroidectomy*: Remove most of the bulk of the gland leaving a normal size thyroid (size of thumb) in tracheo-esophageal groove on both sides taking care not to injure recurrent laryngeal nerve and parathyroid glands. Although postoperative thyroxin is given routinely in these cases, it doesn't prevent recurrence of thyroid nodules.
2. *Total thyroidectomy*: There are chances of recurrence after subtotal thyroidectomy since etiological factors persist. In such cases reoperation is very difficult and hazardous. So another option, especially in younger patients, is total thyroidectomy with preservation of bilateral recurrent laryngeal nerves and at least one parathyroid gland. The patient is put on lifelong thyroxin replacement.
3. *Lobectomy*: If only one lobe of thyroid gland is involved and the other lobe is not palpable, treatment is removal of involved lobe only (Figs 23.10A to C)

Retrosternal Goiter

Lower pole of multinodular goiter may extend behind sternum to form retrosternal goiter. It is mostly seen in men with short neck and strong ribbon muscles. The nodular goiter is sucked in superior mediastinum by negative intrathoracic pressure. Rarely it may arise from ectopic thyroid tissue.

- Mostly asymptomatic and discovered on clinical examination (lower limit of goiter can't be reached).
- Dyspnea, dysphagia and engorged veins of neck and chest wall.



Fig. 23.10A: Multinodular goiter involving left lobe only

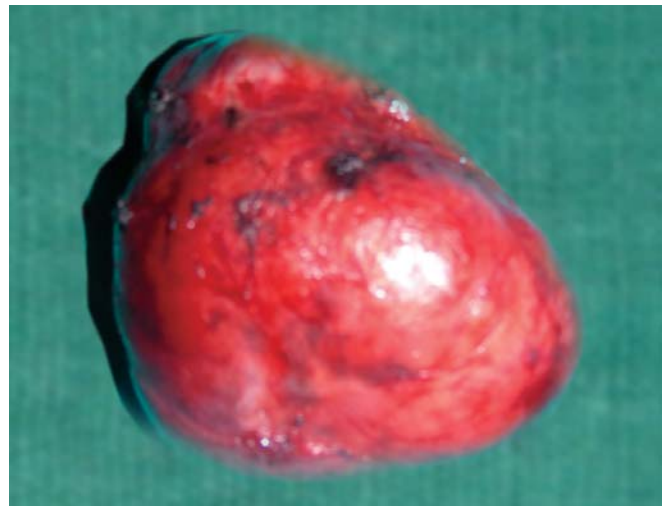


Fig. 23.10B: Left lobectomy specimen



Fig. 23.10C: Left lobectomy cut section

- Pemberton's sign: The neck veins become more prominent when hands are raised above the head and arms touch the ears due to compression of superior vena cava.
- X-ray chest shows soft tissue shadow in superior mediastinum causing tracheal compression and deviation.
- Treatment is surgical resection. Most of the times retrosternal goiter can be mobilized through cervical incision with finger dissection. Hemorrhage is rarely seen since blood supply is coming from the neck. Sometimes, median sternotomy may be required if goiter is stuck behind the sternum.
- In case of associated thyrotoxicosis with retrosternal goiter, antithyroid drugs or radioiodine should not be given because these agents cause edema of thyroid leading to exaggeration of symptoms.

THYROTOXICOSIS

It is a clinical syndrome resulting from excess circulating free T_4 and free T_3 . Its different clinical types are:

I. Diffuse Toxic Goiter

- Also known as **Graves' disease** or **primary thyrotoxicosis** (Box 23.7).
- It is most common cause of thyrotoxicosis (70% cases).
- It is an autoimmune disorder.

Box 23.7: Differences between primary and secondary thyrotoxicosis

Graves' disease	Plummer's disease
1. Young patient	1. Old patient
2. Severe symptoms, rapid onset.	2. Mild symptoms, slow onset.
3. Family history present	3. No family history
4. Diffuse soft and smooth enlargement of thyroid gland (Fig. 23.13)	4. Gland is firm and nodular
5. Bruit present at upper pole of thyroid.	5. Bruit usually absent.
6. Exophthalmos present	6. Exophthalmos absent
7. CNS symptoms	7. CVS symptoms (heart failure, arrhythmia).
8. High recurrence after surgery	8. Low recurrence after surgery

- Production of thyroid stimulating antibodies (TSAb) leads to diffuse hypertrophy and hyperplasia of thyroid gland.
- It involves young females and mostly patients have eye signs.
- Disease is known to have remissions and exacerbations.

II. Toxic Multinodular Goiter

- Also known as **Plummer's disease** or **secondary thyrotoxicosis** (Box 23.7).
- It is mostly seen in older patients with long standing MNG.
- Mostly internodular area is active and nodules are inactive. Rarely nodules may become overactive.
- The clinical features are usually mild and eye signs are absent.

III. Toxic Adenoma

- It is a solitary nodule in the thyroid which is autonomous and overactive.
- Excess release of thyroxine from the toxic nodule leads to decrease in TSH levels by negative feedback mechanism. This in turn leads to suppressed normal thyroid tissue around the nodule that is seen as 'hot spot' on thyroid scan.

IV. Rare Causes

- Thyroiditis
- Metastatic thyroid carcinoma
- Exogenous iodine/thyroid hormone
- Thyrotoxicosis factitia—due to overdose of thyroxine.
- Jod-Basedow's thyrotoxicosis—iodine induced toxic goiter.
- Struma ovarii—ectopic thyroid tissue in malignant ovarian tumor.
- Drugs—amiodarone (antiarrhythmic drug).

CLINICAL FEATURES OF THYROTOXICOSIS (ANY CAUSE)

Symptoms

- Heat intolerance and sweating
- Decreased weight and increased appetite
- Irritability, nervousness
- Diarrhea
- Palpitation
- Oligomenorrhea.



Fig. 23.11: Demonstrating tongue tremors with mouth open and tongue without protrusion



Fig. 23.12: Demonstrating fine finger tremors by looking for paper dance

Signs

- Tremors: Tongue tremors (Fig. 23.11), Finger tremors (Fig. 23.12)
- Warm, moist skin
- Tachycardia (High sleeping pulse rate)
- Atrial fibrillation
- Cardiac failure
- Goiter with bruit
- Lid lag, lid retraction.

Specific to Graves' Disease

- Ophthalmopathy (see thyroid eye disease)
 - ☐ Grittiness
 - ☐ Periorbital edema
 - ☐ Conjunctival edema (chemosis)
 - ☐ Bulging eyes (proptosis)
 - ☐ Diplopia (extraocular muscle involvement)
 - ☐ Impaired vision (optic nerve compression)

Box 23.8: Clinical findings in toxic goiter

Grave's disease	Plummer's disease	Toxic adenoma
Uniform enlargement (butterfly shape)	Irregular enlargement	Circumscribed nodule
Smooth surface	Nodular surface	Smooth surface
Soft consistency	Firm consistency	Firm consistency
May be pulsatile (due to increased vascularity)	Non-pulsatile	Non-pulsatile
Warm	Not warm	Not warm
Bruit heard on auscultation	No bruit	No bruit



Fig. 23.13: Graves' disease

- Pretibial myxoedema (nonpitting edema, thick skin of pretibial region)
- Thyroid acropathy (finger clubbing)
- Thyroid myopathy (weakness of proximal limb muscles).

Comparison of clinical findings in toxic goiter due to various causes is given in Box 23.8.

DIAGNOSIS FOR THYROTOXICOSIS

- Investigations are same as for goiter (already described).
- Most of the times, diagnosis of thyrotoxicosis is easily made on clinical findings. In doubtful cases thyrotoxicosis should always be suspected in following situations:
 - ☐ Children with behavior problems, CNS symptoms.

- ☐ Elderly with cardiac problems (arrhythmia, CHF)
- ☐ Unexplained diarrhea and weight loss.
- On investigations, TSH is decreased and T_3 , T_4 levels are high.
- Levels of TSAb are high in Graves' disease.
- On thyroid scan, hot nodule is seen in toxic adenoma, diffuse increase in uptake is seen in Graves' disease and patchy uptake is seen in toxic MNG.

TREATMENT

There are three modalities of treatment, namely antithyroid drugs, surgery and radioiodine (Box 23.9). The choice of treatment is given in Box 23.10

Box 23.9: Comparison of various treatment modalities for thyrotoxicosis

	Advantages	Disadvantage
Antithyroid drugs	No risk of complications of surgery or radioiodine therapy.	Prolonged treatment. Recurrence after stopping treatment. Drug toxicity (agranulocytosis).
Surgery	Rapid and high cure rate.	Surgical complications. Recurrent Laryngeal N. injury. Hypoparathyroidism. Recurrent thyrotoxicosis. Postoperative hypothyroidism.
Radioiodine	Easy to administer. No surgery or prolonged drug therapy.	Permanent hypothyroidism. Genetic mutation.

Box 23.10: Choice of treatment in thyrotoxicosis

Graves' disease	After 40 years of age: ☐ Radioiodine Below 40 years of age: ☐ Antithyroid drugs for small goiter ☐ Surgery for large goiter
Toxic nodular goiter	Surgery
Toxic adenoma	Surgery Radioiodine (after 40 years of age)
Proptosis	Sudden control of toxicosis by surgery or radioiodine may induce malignant exophthalmos. So, antithyroid drugs should be given initially to stabilize proptosis for about 6 months.

Antithyroid Drugs (Box 23.11)

- Carbimazole and propylthiouracil are the main drugs.
- These interfere with T_3 and T_4 synthesis by inhibiting oxidation and coupling.
- Initial dose of carbimazole is 40 mg/day (10 mg 6 hrly) which can be gradually reduced to 20 mg/day (10 mg 12 hrly) to maintain euthyroid state.
- Course of therapy ranges from 6 months-2 years during which time patient generally goes into remission.
- Side effects are skin rash and agranulocytosis that usually manifests as sore throat.
- In patients with mild symptoms, propranolol can be used in dosage of 10 mg two to three times a day. It is a beta blocker and blocks peripheral receptors for thyroxine. It does not reduce serum levels of thyroxine.

Box 23.11: Antithyroid drugs

Drugs and dosage	Mechanism of action	Remarks
Carbimazole (10 mg 6 hrly)	Inhibits oxidation and coupling	Side effects— agranulocytosis (sore throat) and skin rash
Propyl thiouracil (200 mg 8 hrly)	Inhibits oxidation	Given to patients developing agranulocytosis with carbimazole.
Propranolol (10 mg 8 hrly)	Blocks peripheral receptors	Continue after surgery for thyrotoxicosis since it doesn't reduce serum thyroxin levels. Side effects—CHF and bronchial asthma
Lugol's iodine (potassium iodide) (5 mg 8 hrly)	Reduces vascularity of thyroid gland	Doubtful role, Bitter taste and Started 10 days before surgery.

- Amiodarone is antiarrhythmic drug and contains iodine. Its concomitant use can worsen thyrotoxicosis.
- In toxic MNG or toxic adenoma, antithyroid drugs cannot cure autonomous and overactive thyroid tissue and recurrence occurs on stopping the drugs. So, definitive treatment is surgery or radiotherapy.
- In Graves' disease, 50% cases may go into prolonged remission following antithyroid drugs. So medical treatment is the primary treatment.

Surgery

- Make the patient euthyroid with antithyroid drugs before surgery.
- Potassium iodide (5 mg TDS) may be added about 10 days prior to surgery to cause regression in size of the gland.
- Toxic adenoma—do lobectomy.
- MNG, large gland, relapse after medical treatment—do subtotal thyroidectomy (about 5 gm gland left on each side).
- It is better to do more removal of gland because postoperative hypothyroidism is easier to treat rather than recurrent thyrotoxicosis.

Radioiodine

Sodium iodide (Na^{131}I) is given orally which is rapidly taken up by the thyroid. Dose is 150 microcurie/gm of thyroid. Beta emissions cause progressive death and stromal fibrosis. It is used in patients above 40 years of age. The treated patient gradually becomes euthyroid and many will develop hypothyroidism over months or years.

Its *indications* are:

- Relapse after medical treatment or surgery.
- Contraindication to medical treatment or surgery.

Its *contraindications* are:

- Pregnancy and lactation.
- Usually not given in children due to theoretical risk of carcinogenesis.

THYROID NEOPLASMS

Benign Tumors

- **Follicular adenoma** presents as solitary thyroid nodule.

- FNAC is unable to differentiate follicular adenoma from follicular carcinoma. The two can be differentiated on the basis of capsular invasion and vascular invasion that can only be seen on histopathological examination.
- Treatment is lobectomy.
- There is no term like papillary adenoma since all papillary tumors are malignant.

Malignant Tumors

Malignant tumors of thyroid are uncommon and account for only 1% of all malignancies. However, these are the most common malignant endocrine tumors (Box 23.12).

Box 23.12: Malignant thyroid tumors—incidence

• Papillary carcinoma	60%
• Follicular carcinoma	20%
• Anaplastic carcinoma	10%
• Medullary carcinoma	5%
• Others (lymphoma, metastatic, etc.)	5%

Etiology

1. *Irradiation*: Radiotherapy to neck (e.g. for lymphoma) has been implicated in papillary carcinoma.
2. *Endemic goiter*: Long standing MNG may change to follicular carcinoma.
3. *Hashimoto's thyroiditis*: It may lead to malignant lymphoma.

Pathology

Types of malignant tumors are:

1. Differentiated thyroid carcinoma
 - Papillary carcinoma
 - Follicular carcinoma
2. Undifferentiated (anaplastic) carcinoma
3. Medullary carcinoma
4. Lymphoma.

Clinical Features

- Thyroid cancer usually presents as a lump in the neck which clinically may be a solitary nodule or multinodular goiter.
- About 10% of thyroid nodules are malignant.
- Dominant nodule in MNG has same cancer risk as solitary nodule.

- A thyroid nodule should be viewed with suspicion if it has following features:
 - ☐ Family history of thyroid cancer.
 - ☐ History of neck irradiation in past.
 - ☐ Age <15 years or > 65 years, especially male patient.
 - ☐ Recent origin.
 - ☐ Rapid increase in size.
 - ☐ Hoarseness of voice.
 - ☐ Firm, fixed, irregular nodule in thyroid.
 - ☐ Along with enlarged cervical lymph nodes.

DIFFERENTIATED THYROID CARCINOMA

i. Papillary Carcinoma

- Most common form of thyroid carcinoma (60-80%).
- Most papillary tumors are mixture of papillary and follicular neoplasm, but they are treated as papillary carcinoma.
- Most common in children and young adults.
- Histologically, papillary projections are seen with calcified areas (psammoma bodies). Cells contain pale and empty looking nuclei (Orphan Annie eyed nuclei).
- Tumor is multifocal involving one or both lobes due to rich intrathyroidal lymphatic plexus.
- Metastasis occurs commonly by lymphatic spread to cervical lymph nodes (Fig. 23.14). The blood born metastasis is unusual. However, lymph node involvement does not worsen the prognosis (Box 23.13A).

Box 23.13A: Papillary carcinoma—metastatic cervical lymph nodes

- Lower deep cervical nodes are usually involved on the side of lesion.
- Firm or cystic in consistency.
- Mobile or fixed.
- Only cervical nodes may be palpable with non-palpable thyroid in occult tumor (lateral aberrant thyroid).
- Lymph node metastasis does not worsen the prognosis.
- Modified radical neck dissection is the treatment of choice.
- Berry picking (removal of only enlarged lymph nodes) is not practised these days.



Fig. 23.14: Multiple cervical lymph nodes appearing two years after thyroidectomy for papillary carcinoma thyroid



Fig. 23.15: Left supraclavicular lymph node mass—metastatic deposits from occult papillary carcinoma thyroid

- The tumor not palpable clinically and detected on histopathology (up to 1.5 cm) is called occult tumor. Clinically it may present with only cervical lymphadenopathy (lateral aberrant thyroid) (Fig. 23.15).
- The preoperative diagnosis is usually made by FNAC of thyroid nodule and/or enlarged cervical lymph node.
- A patient is considered low/high-risk based on absence or presence of 'AMES criteria' (Box 23.13B).

Box 23.13B: 'AMES criteria' for differentiated thyroid carcinoma (Papillary and Follicular)

The prognosis is poor in:

A: Old age (>50 years female, >40 years male)

M: Presence of distant metastasis

E: Extra thyroidal extent (tumor extending outside the capsule of thyroid).

S: Size >5 cm.

CASE SUMMARY

60 years old male presented with painless, progressively increasing swelling in left supraclavicular region for the last one year. There were no associated symptoms. On examination, there was a soft, cystic mass in left supraclavicular region. Its lower limit could not be reached (Fig. 23.15). FNAC done twice was inconclusive. It was provisionally diagnosed as cystic hygroma and excision was planned. Ultrasound examination was performed to see the lower extent of mass and a small hypoechoic lesion was incidentally picked up in left lobe of thyroid during ultrasound. Ultrasound guided FNAC of the lesion made the diagnosis of papillary carcinoma thyroid. Patient underwent total thyroidectomy with left MRND. Isotope scan was performed six weeks after surgery and there was no residual tumor. Patient was put on tablet Eltroxin and is on regular follow-up.

Learning point: It is a classical example of occult primary in thyroid with metastasis in cervical lymph nodes (lateral aberrant thyroid).

ii. Follicular Carcinoma

- Constitute 10-20% of all thyroid cancers.
- Higher incidence in endemic areas (Fig. 23.16).
- It is not multifocal and lymph node involvement is rare.
- Presence of capsular and vascular invasion differentiates it from follicular adenoma.
- Metastasis is blood borne to lungs and skeleton. Metastasis is functional and takes up radioiodine (Box 23.13C).
- FNAC is not helpful in making diagnosis of follicular carcinoma because FNAC cannot identify presence of capsular and vascular invasion. Hence, the diagnosis of follicular carcinoma is established on excision biopsy (lobectomy).



Fig. 23.16: Follicular carcinoma developing in long standing multinodular goiter

Box 23.13C: Follicular carcinoma—skeletal metastasis

- Due to hematogenous spread.
- Grows very rapidly.
- Involves flat bones (skull, sternum, ribs, vertebrae) due to presence of red marrow.
- Presents as
 - ☐ Pulsatile mass
 - ☐ Pathological fracture.
- X-ray shows osteolytic lesion.
- Increased alkaline phosphatase levels.
- Treatment
 - ☐ Palliative (Radioiodine/RT)

- The patients are divided into low/high-risk groups based on absence or presence of 'AMES criteria' (Box 23.13B).
- **Hurthle cell neoplasm** is a variant of follicular carcinoma and is rich in oxyphilic cells. It has unpredictable behavior because benign and malignant neoplasm is difficult to distinguish. Moreover, it does not take up radioactive iodine. Hence, total thyroidectomy is the treatment of choice. Comparison between two types of differentiated thyroid carcinoma is shown in Box 23.14.

Treatment of Differentiated Thyroid Cancer

The strategy of surgical treatment for differentiated thyroid cancer is decided on the basis of low or high-

Box 23.14: Differentiated thyroid carcinoma

	<i>Papillary carcinoma</i>	<i>Follicular carcinoma</i>
Incidence	60% (Most common)	20%
Age	More in young adults	More in middle age
Sex	More in males	More in females
Premalignant condition	Neck irradiation	Endemic goiter
Pathology	Multifocal tumor Psammoma bodies Orphan Annie eyed nuclei	Not multifocal Presence of capsular and/or vascular invasion.
FNAC	Makes diagnosis	Diagnosis not on FNAC. Tissue biopsy required for diagnosis
Metastasis	Lymphatic	Hematogenous
TSH dependence	Yes	Yes
Radioiodine uptake	Yes	Yes

risk group patient (AMES criteria). In low-risk group patients, hemithyroidectomy (lobectomy + isthmusectomy) is sufficient, while high-risk group patients require total thyroidectomy. Points favoring hemithyroidectomy vs total thyroidectomy are given in Box 23.15.

In case, patient presents with a solitary thyroid nodule that is suspected to be malignant, the management outlines are given in Box 23.16.

i. Papillary Carcinoma

Following are the principles of surgical treatment:

- Patient with 1 cm tumor with no palpable lymph nodes
 - ☐ hemithyroidectomy
- Patient with tumor > 1 cm
 - ☐ Total thyroidectomy
- Patient with multifocal or bilateral disease, node positive
 - ☐ Total thyroidectomy.
- In patients with enlarged cervical nodes, modified radical neck dissection is done where internal jugular vein, accessory nerve and sternomastoid muscles are preserved.

ii. Follicular Carcinoma

Following are the principles of surgical treatment:

- All follicular neoplasms involving one lobe are initially treated with hemithyroidectomy.
- Lesions with no capsular and vascular invasion—no further treatment.

Box 23.15: Differentiated thyroid cancer—hemithyroidectomy vs total thyroidectomy

Total Thyroidectomy: Points in favor

1. Multifocal disease involving both lobes.
2. Reduced chances of local recurrence.
3. Ablation with radioiodine is facilitated.
4. Low morbidity in experienced hands.

Hemithyroidectomy: Points in favor

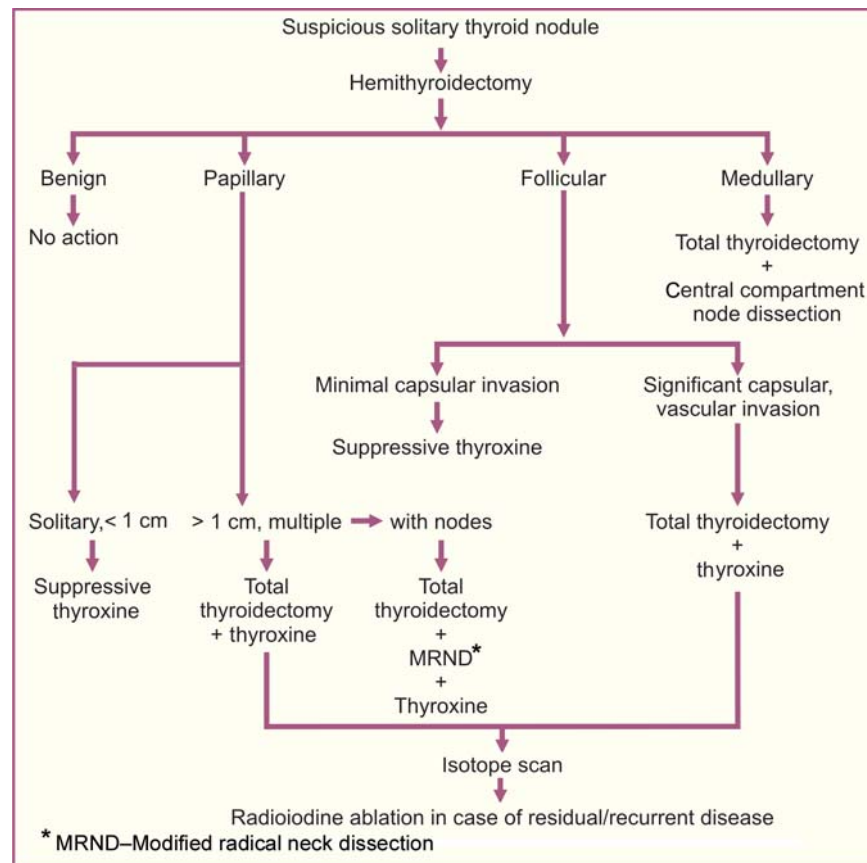
1. Significance of microfoci in opposite lobe is uncertain.
2. Local recurrence infrequent after hemithyroidectomy.
3. If indicated, remaining thyroid tissue can be ablated with radioiodine.
4. Higher incidence of hypoparathyroidism after total thyroidectomy.

- Lesions with minimal capsular invasion—suppressive thyroxine therapy.
- Lesions with vascular invasion or local fixity—total thyroidectomy.
- Hurthle cell tumor—total thyroidectomy.

Postoperative Management

- After surgery, thyroxine replacement is given to prevent hypothyroidism and to suppress TSH since differentiated tumors are TSH dependent.
- In differentiated thyroid tumors, radioactive iodine scanning is done after surgery because these tumors take up iodine.
- Isotope scanning is not needed in low-risk patients where hemithyroidectomy is sufficient.

Box 23.16: Suspicious solitary thyroid nodule



- In high-risk patients (extra-thyroidal tumor, positive nodes, distant spread), scanning is done six weeks after surgery. Scanning is not able to pick up distant metastasis if substantial thyroid tissue is left in place. If residual thyroid tissue and/or metastases are picked upon scan, then the patient is given a therapeutic dose of radioiodine.
- In preparation for scan, allow endogenous TSH to increase by stopping thyroxine for 4 weeks before the scan.
- After ablation with radioiodine, the patient can be followed-up by monitoring serum thyroglobulin levels (tumor marker) and a rising level will indicate residual or recurrent disease.

ANAPLASTIC CARCINOMA

It is an uncommon tumor and affects elderly patients (>60 years) and has higher incidence in areas of endemic goiter. Many cases arise from pre-existing, undiagnosed well-differentiated tumors. There is long

standing history of goiter that suddenly starts enlarging rapidly. Tumor rapidly infiltrates adjoining structures and metastasizes by blood and lymphatics. Pressure symptoms like dyspnea, dysphagia and hoarseness of voice predominate. The tumor carries a very poor prognosis and most patients die within one year. This feature emphasizes need for adequate treatment of well-differentiated tumors and full evaluation of all goiters.

FNAC is helpful in making the diagnosis.

Thyroidectomy is usually not possible due to wide spread disease. Treatment is palliative for relief of symptoms. For airway obstruction, tracheostomy should be avoided and tracheal pressure can be relieved by division of isthmus. External beam radiotherapy helps in local control (Box 23.17).

MEDULLARY CARCINOMA

- It is a rare tumor that arises from parafollicular or C-cells of thyroid gland (neural crest origin).

Box 23.17: Anaplastic carcinoma thyroid

- Incidence—10%
- Elderly age—> 60 years
- High incidence in endemic goiter
- Rapidly growing
- Local infiltration
- Hematogenous and lymphatic spread
- Diagnosis on FNAC
- Doesn't take up radioiodine
- Not TSH dependent
- Treatment—palliative (isthmusectomy, RT)
- Poor prognosis

- It is not TSH dependent and does not take up radioiodine.
- Its hormone marker is calcitonin.
- It can present in sporadic and familial form.
- Sporadic presents as unilateral tumor while familial is almost always bilateral.
- In familial form (MEN type II) it is associated with other endocrine tumors of adrenal and parathyroid gland. It is essential to exclude co-existing pheochromocytoma in these cases before planning surgery (Box 23.18).
- The **clinical presentation** is usually as thyroid swelling with enlarged cervical lymph nodes. Patient may complain of diarrhea due to hormones and peptides secreted by the tumor. Distant spread may occur to lungs, liver and bones.
- **Diagnosis** is made by FNAC and raised serum calcitonin levels. Microscopically, hyperchromatic

Box 23.18: Multiple endocrine neoplasia (MEN) syndrome

- **MEN Type I**
 - Pituitary adenoma
 - Parathyroid adenoma
 - Pancreatic adenoma
- **MEN Type IIa**
 - Parathyroid adenoma
 - Pheochromocytoma
 - Medullary carcinoma thyroid
- **MEN Type IIb**
 - Same as Type IIa +
 - Neuromas of tongue, lips, eyelids

Box 23.19: Medullary carcinoma thyroid

- Rare tumor (5%).
- Origin—parafollicular cells.
- Microscopy—amyloid stroma.
- Secretes calcitonin (tumor marker).
- Secretes hormones and peptides (diarrhea).
- Sporadic or familial.
- In familial, investigate for parathyroid and adrenal tumors.
- Spread—lymphatic, blood.
- Not TSH dependent.
- Does not take up radioiodine.
- Diagnosis by FNAC.
- Surgery is the only modality of treatment.
- Prognosis depends on lymph node metastasis.

nucleus with amyloid stroma are characteristic features.

- **Treatment** is primarily surgical. Total thyroidectomy and central compartment lymph node clearance is recommended for all patients. In case lateral lymph nodes are involved, modified radical node dissection is required. Prognosis and survival depends on presence or absence of lymph node metastasis.
- In familial cases, genetic screening for the RET oncogene mutation can identify cases who will develop medullary carcinoma later in life. Prophylactic total thyroidectomy should be considered in such cases at the age of 5-7 years.
- Summary of medullary carcinoma thyroid is given in Box 23.19.

MALIGNANT LYMPHOMA

It tends to arise in pre-existing long standing Hashimoto's thyroiditis. It is more common in elderly women. It may present as dominant nodule, multinodular goiter or a rapidly growing neck mass with accompanying lymph node enlargement.

The diagnosis can be made on FNAC but core biopsy is necessary to allow immunocytochemical subtyping. Treatment is by radiotherapy and chemotherapy, thyroidectomy is not indicated.

SOLITARY THYROID NODULE

- Well-circumscribed, single nodule is palpable in thyroid while remaining gland is not palpable (Fig. 23.17).



Fig. 23.17: Solitary thyroid nodule of right lobe

- In 50% cases, underlying pathology is multinodular goiter having one dominant nodule while remaining nodules are microscopic, hence not palpable.
- In remaining cases, the causes are adenoma, carcinoma (papillary or follicular) and thyroiditis (Box 23.20).
- In case of toxic adenoma (autonomous nodule), it is almost never caused by malignant nodule.
- FNAC is the investigation of choice for determining underlying pathology (Box 23.21).

Box 23.20: Solitary thyroid nodule—causes

- Part of MNG.
- Toxic adenoma.
- Adenoma.
- Carcinoma.
- Cyst.
- Thyroiditis.

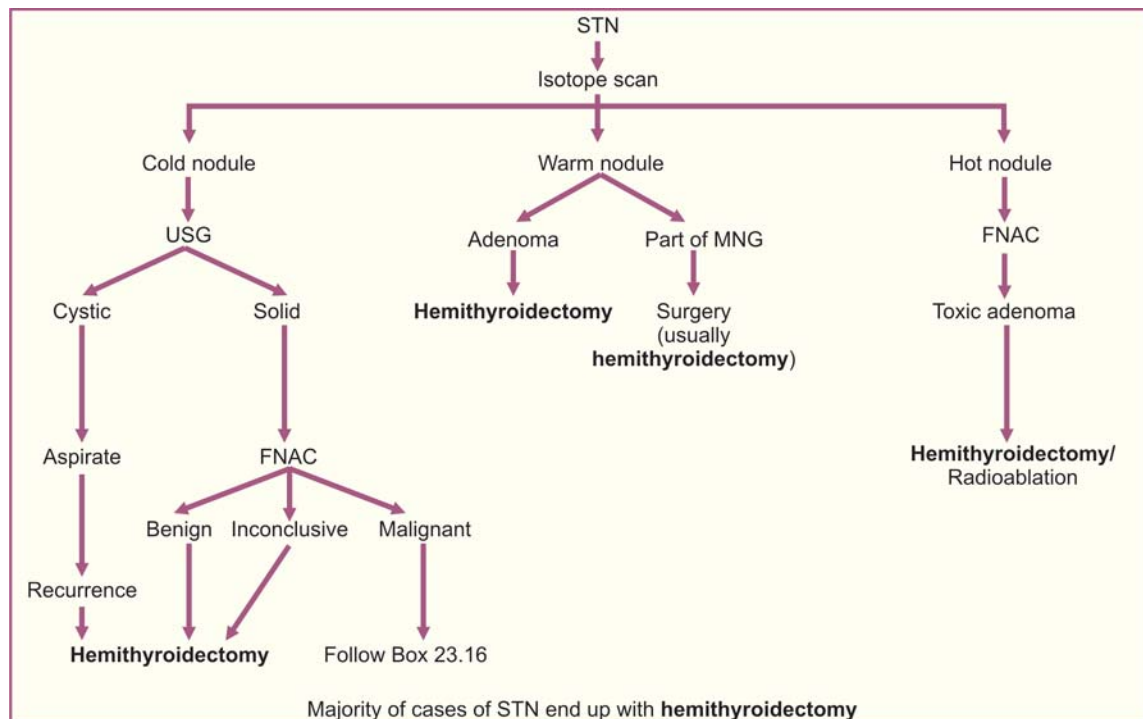
Box 23.21: Solitary thyroid nodule—investigations

- T₃, T₄, TSH.
 - Isotope scan (hot/warm/cold nodule).
 - USG (solid/cystic).
 - FNAC.
 - Excision biopsy (hemithyroidectomy).
- If FNAC is inconclusive and the nodule is suspicious it should be subjected to hemithyroidectomy (see Box 23.16 and Box 23.22).

THYROIDITIS

It is a group of heterogenous disorders where common feature is inflammation of thyroid gland. It mainly affects women and clinical course may be acute, subacute or

Box 23.22: Solitary thyroid nodule (STN)—management



chronic. Patients may present with euthyroidism, transient hyperthyroidism or hypothyroidism and sometimes all three thyroid states may occur during the course of disease.

Hashimoto's Thyroiditis (Chronic Autoimmune or Lymphocytic Thyroiditis)

It is most common cause of thyroiditis.

Histologically, there is diffuse lymphocytic infiltration, follicular destruction, colloid depletion and fibrosis.

Clinically, most patients present with a painless, diffuse goiter of variable consistency (rubbery, firm or hard) depending on the cellularity and the degree of fibrosis.

In patients of Hashimoto's thyroiditis, sudden growth of goiter should raise the suspicion of malignant change (lymphoma, papillary carcinoma). Initially, patients may have transient hyperthyroidism but ultimately, hypothyroidism occurs in most patients.

Diagnosis is mostly made on FNAC. Thyroid antibody titers are raised in most cases.

Treatment is with thyroxine replacement therapy (0.2 mg/day) in patients with hypothyroidism. If goiter is increasing in size, steroid therapy may help. However, increasing goiter should raise the suspicion of malignant change. In large goiter causing discomfort and cosmetic deformity, thyroidectomy is indicated.

Granulomatous Thyroiditis (Subacute or de Quervain's Thyroiditis):

It is most common cause of painful thyroid. It is often preceded by upper respiratory tract viral infection. The

patient complains of fever, malaise and painful thyroid swelling. Transient hyperthyroidism occurs in half the cases. The condition is self-limiting and resolves in about 8 weeks.

ESR is raised and thyroid antibodies are usually absent. If diagnosis is in doubt, it can be confirmed with FNAC. Treatment is symptomatic and consists of NSAIDs for pain relief. In case of severe pain, steroids may be given.

Reidel's Thyroiditis

It is very rare condition of unknown etiology. There is dense invasive fibrosis of thyroid that extends beyond thyroid gland into surrounding tissues.

It may be associated with retroperitoneal and mediastinal fibrosis. The patient presents with rapidly increasing thyroid with symptoms of tracheal and esophageal compression.

On examination, thyroid is uniformly enlarged, 'woody hard' and fixed. Biopsy is usually needed to exclude malignancy. Isthmusectomy may be done to relieve pressure symptoms on trachea and esophagus.

Comparison between three types of thyroiditis is given in Box 23.23.

THYROIDECTOMY—OPERATIVE STEPS

- Patient is operated in supine position under general anesthesia.
- A small sandbag is placed between the shoulders to extend the neck and head is supported upon a ring.
- A 'collar incision' (neckline incision) is made in the neck along skin creases 1" above the sternum extend-

Box 23.23: Thyroiditis

	<i>Hashimoto's</i>	<i>Granulomatous</i>	<i>Riedel's</i>
Etiology	Autoimmune disease	Viral infection	Unknown
Age	Middle age	Young age	Old age
Microscopy	Lymphocytic infiltration, Follicular destruction	Inflammatory cells	Dense fibrosis
Symptoms	Painless thyroid swelling	URI, fever, painful thyroid swelling	Rapidly increasing swelling with symptoms of compression
Toxicity	Initially mild hyperthyroidism followed by hypothyroidism	Initially hyperthyroidism, later normal	Hypothyroidism
Investigations	Antithyroid antibodies	ESR ↑	—
Treatment	Thyroxin, surgery	Symptomatic	Thyroxin, Isthmusectomy
Premalignant	Yes (lymphoma)	No	No

ing between the lateral borders of sternomastoid muscles.

- The flaps of skin and platysma are raised; upper flap upto thyroid cartilage and lower flap upto sternum.
- The investing layer of deep fascia is incised vertically in midline, the ribbon muscles are retracted laterally and the thyroid gland is exposed.
- The middle thyroid vein is ligated and divided first and the lobe is delivered.
- Superior thyroid vessels are ligated and divided at upper pole taking care not to injure superior laryngeal nerve.
- Inferior thyroid vessels are ligated and divided at lower pole taking care not to injure recurrent laryngeal nerve.
- Current practice is to ligate individual branches of inferior thyroid artery after it has given supply to the parathyroid glands so as to avoid ischemic damage to the parathyroid glands.
- The thyroid lobe is completely mobilized and divided at isthmus (Lobectomy).
- In total thyroidectomy same procedure is repeated on the other side taking care to preserve at least one parathyroid gland.
- Hemostasis is achieved and the wound is closed after putting a closed suction drain in the thyroid bed.

COMPLICATIONS OF THYROIDECTOMY

Nerve Damage

External branch of superior laryngeal nerve is the most commonly damaged nerve. Injury results in loss of vocal cord tension leading to decreased pitch of voice (important for singers). This damage often remains unrecognized.

Recurrent laryngeal nerve damage affects motor supply to vocal cords leading to vocal cord palsy. Vocal cords allow phonation, protect airways and facilitate coughing. Hence, recurrent laryngeal nerve should be identified and protected in all cases (Fig. 23.18).

Unilateral recurrent laryngeal nerve injury causes hoarseness of voice and reduced force of coughing. In most cases, there is partial injury (neuropraxia) and recovery occurs in 3 weeks time. Patients with permanent injury and no improvement may improve with teflon injection in vocal cords and speech therapy.

Bilateral recurrent laryngeal nerve injury leaves both the vocal cords in paramedian position. It is because of

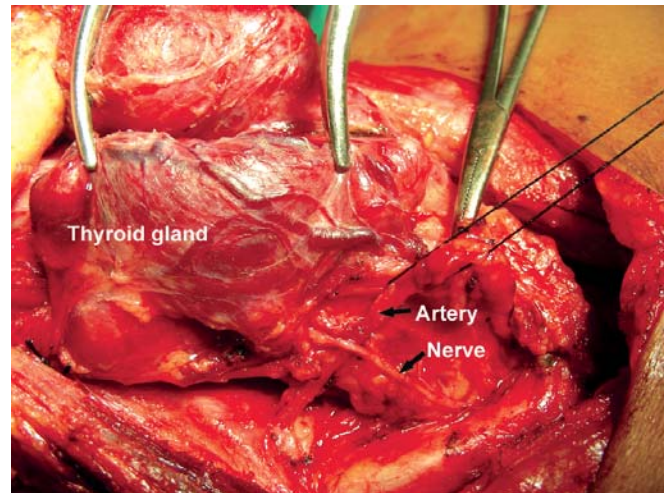


Fig. 23.18: Thyroidectomy—showing recurrent laryngeal nerve and inferior thyroid artery

unopposed adducting action of cricothyroid muscles that are supplied by superior laryngeal nerves (also see Chapter 16: Diseases of Larynx). Hence, patient develops respiratory obstruction on removal of endotracheal tube after surgery. In such situation, airway is restored by doing tracheostomy. Wait for 6 months to 1 year till recovery may occur. After 1 year, recovery is unlikely and treatment is vocal fold lateralization procedure in form of

Arytenoidectomy—removal of arytenoid cartilage
or

Lateral arytenoidpaxy—suturing arytenoid cartilage laterally.

Postoperative Bleeding

It causes laryngeal compression and respiratory obstruction. Treatment is immediate re-exploration under GA, evacuation of clots and suture ligation of bleeding vessels.

Hypocalcemia

It occurs after bilateral thyroid surgery due to inadvertent vascular injury to parathyroid glands. Treatment is injection calcium gluconate 10 ml intravenous slowly followed by oral calcium and vitamin D supplement.

Hypothyroidism

It can occur after bilateral thyroid surgery. Monitoring is done with thyroid function tests and treatment is with thyroxine.

Thyrototoxic Crisis

It usually occurs if thyroid surgery is performed in a patient with uncontrolled thyrotoxicosis. Patient presents with acute manifestations of thyroid over activity in form of high grade fever, sweating, hypotension, tachycardia and prostration.

Treatment is with intravenous fluids, cold sponging, intravenous propranolol, steroids and neomercazole.

Scarring and Keloid Formation

It is especially seen in dark skin persons.

Wound Infection

It is seen rarely.

Tracheomalacia

Large goiter may cause prolonged tracheal compression leading to tracheomalacia. After surgery, patient develops tracheal collapse and respiratory obstruction requiring tracheostomy.

ECTOPIC THYROID

Some residual thyroid tissue may remain along thyroglossal tract (Fig. 23.19). It may be lingual, cervical or intrathoracic.

Lingual thyroid forms rounded swelling at back of tongue at foramen caecum. It may present with dysphagia, respiratory obstruction, impaired speech and bleeding.

Treatment is full replacement dose of thyroxine that makes it smaller.

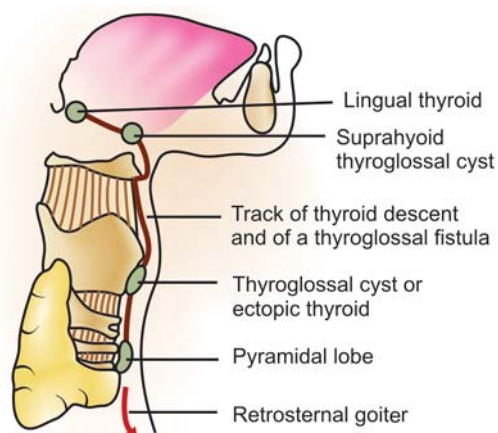


Fig. 23.19: Descent of thyroid

Sometimes excision is required for large swelling. However, thyroid scan should be done to confirm the presence of normal thyroid tissue before excision since lingual thyroid may represent only thyroid tissue in the body.

THYROGLOSSAL CYST

It is a cystic swelling in the midline of neck anywhere from foramen caecum in tongue to thyroid isthmus (location of thyroglossal tract). Most common location is subhyoid (Fig. 23.20). The cyst moves upwards on swallowing as well as on protrusion of tongue due to attachment of the tract to foramen caecum (Figs 23.21 and 23.22).

Due to presence of lymphoid tissue it may get infected and appears like an abscess. This abscess may rupture or got incised leading to formation of thyroglossal fistula.



Fig. 23.20: Thyroglossal cyst



Fig. 23.21: Thyroglossal cyst with tongue inside oral cavity; chin has been stabilized with a finger to prevent jaw movements



Fig. 23.22: Thyroglossal cyst moves up on tongue protrusion



Fig. 23.23: Thyroglossal fistula

Thyroglossal fistula is lined with columnar epithelium, discharges mucus and gets recurrent attacks of infection (Fig. 23.23).

Treatment is excision of thyroglossal cyst/ fistula along with thyroglossal tract including central part of hyoid bone as well as central core of lingual muscle (**Sistrunk's operation**).

THYROID EYE DISEASE

It is due to infiltration of intraocular muscles with T- cells due to immune mechanism. It leads to bulging of eye balls (exophthalmos). It is mostly seen in hyperthyroid patients. For examination, stand behind the patient and look at the superciliary arch by tilting the patient's head backwards (**Naffziger's method**). In normal case, eye-balls are not seen while in exophthalmos, eyeballs protrude outside (Fig. 23.24B).

There is no relation between severity of hyperthyroidism and ocular involvement. Patient feels ocular irritation in form of grittiness, watering. There is conjunctival congestion, edema followed by ulceration that can even lead to blindness. In severe and progressive form, it is called as *malignant exophthalmos* and eye may be destroyed (Box 23.24).

Various eye signs seen in hyperthyroidism are:

(i) Stelwag's Sign

It is the retraction of upper eyelid due to spasm of *levator palpebrae superioris* muscle which has sympathetic



Fig. 23.24A: Stelwag's sign—upper limbus is visible



Fig. 23.24B: Naffziger's method of excluding minor bulging of eyeballs

Box 23.24: Thyroid eye disease (exophthalmos)

- Feature of Grave's disease.
- Proptosis due to retrobulbar cell deposition.
- Lid retraction due to smooth muscle spasm.
- Naffziger's method of examination.
- Infrequent blinking of eyes.
- Sclera visible above upper limbus.
- Neglected case – malignant exophthalmos.

innervation. It is most reliable sign. It results in widening of palpebral fissure so that sclera becomes visible above upper limbus (sclero-corneal junction)* (Fig. 23.24A).

*Visible sclera above upper limbus is also called '**Dalrymple's sign**'.

(ii) Von Graefes' Sign

Lid lag on down gaze due to spasm of upper lid.

(iii) Joffroy's Sign

Absence of wrinkling of forehead on upward gaze because field of vision is increased due to exophthalmos.

(iv) Moebius Sign

Loss of convergence of eyeballs due to muscle paresis.

Treatment

- It is symptomatic.
- Artificial tears, sleeping propped up and lateral tarsorrhaphy help in protecting the eye.
- Wearing dark glasses.
- Diuretics to decrease retrobulbar edema.
- High doses of oral prednisolone.
- If eye is in danger, surgical decompression is needed.

CLINICAL EXAMINATION OF THYROID GLAND

History

- **Swelling:** History of swelling in thyroid region.
 - ☐ Duration—
 - i. Long duration in MNG
 - ii. Short duration in malignancy.
 - ☐ Progress—
 - i. Slowly progressive—MNG
 - ii. Rapidly progressive—malignancy.
 - iii. Sudden painful increase in size—hemorrhage in MNG.
 - iv. Increase and decrease in size in between—thyroiditis.
- **Pain:**
 - ☐ Painless swelling—MNG, malignancy.
 - ☐ Painful swelling—thyroiditis.
- **Fever:** It is a feature of thyroiditis.
- **Local pressure symptoms:** These are usually seen in long standing large MNG, retrosternal goiter and infiltrating carcinoma. These are:
 1. Dyspnea: Due to pressure on trachea.
 2. Dysphagia: Due to pressure on esophagus.
 3. Hoarseness of voice: Due to pressure on recurrent laryngeal nerve.

- **Symptoms of thyrotoxicosis:** These are seen in hyperthyroidism.

1. CNS symptoms—primarily in Grave's disease.
 - a. Insomnia
 - b. Restlessness.
 - c. Anxiety.
 - d. Tremors.
 - e. Heat intolerance.
 - f. Preference to cold.
 - g. Eye prominence.
2. CVS symptoms—primarily in secondary thyrotoxicosis.
 - a. Palpitation.
 - b. Chest pain.
 - c. Dyspnea on exertion.
 - d. Edema of feet.
3. GIT symptoms—more in Graves disease
 - a. Diarrhea.
 - b. Increased appetite.
 - c. Decreased weight.
4. Menstrual symptoms—oligomenorrhea.

- **Symptoms of hypothyroidism:**

- a. Tiredness (lethargy).
- b. Decreased appetite.
- c. Weight gain.
- d. Constipation.
- e. Cold intolerance.
- f. Facial puffiness.
- g. Hoarseness of voice.

- **Symptoms of metastasis in thyroid malignancy:**

1. Multiple neck swellings on either side of midline swelling—lymph node metastasis.
2. Cough, hemoptysis and chest pain—suggestive of pulmonary metastasis.
3. Bone pains, bone swelling, fracture following trivial trauma—bony metastasis.
4. Jaundice—liver metastasis.

- **Past history:**

1. History of neck irradiation during childhood (papillary carcinoma thyroid).
2. History of drugs, radioiodine therapy in case of thyrotoxicosis.

- **Family history:**

1. History of goiter in family members is suggestive of dysshormonogenesis.

2. In endemic goiter, thyroid swelling is seen in neighbors as well, apart from family members.
3. Familial form of medullary carcinoma thyroid occurs in family members.

General Physical Examination

- Look at the general appearance. The anxious and agitated look in a thin built patient is suggestive of hyperthyroidism while the hypothyroid patient is obese, slow and lethargic.
- Hold the hands of the patient. Palms are moist and sweaty in thyrotoxicosis.
- Feel the pulse. Tachycardia and irregular pulse is seen in thyrotoxicosis while there may be bradycardia in hypothyroidism.
- Look for the fine tremors by asking the patient to outstretch palms and fingers. The fine tremors can be appreciated by placing a thin sheet of paper on outstretched fingers (see Fig. 23.12).
- Look for tongue tremors by asking patient to open mouth without protruding the tongue (see Fig. 23.11).
- Look for eye signs (See above: Thyroid eye disease).

Local Examination of Neck

- Look for the swelling in the region of thyroid gland and ask the patient to swallow. All thyroid swellings move up on deglutition (Box 23.25) (see Figs 23.6 and 23.7).
- In case the patient is obese and short necked, ask the patient to extend her neck backwards forcefully while pressing the occiput against resistance of her clasped hands (Pizillo's method). It makes the thyroid gland more prominent and easily visible (Fig. 23.25).
- Ask the patient to open the mouth and protrude the tongue. If the swelling moves up on protrusion of tongue, it is likely to be thyroglossal cyst (see Figs 23.20 to 23.22).
- Feel the swelling from front and see for local temperature, tenderness, size, shape, surface, margins and its consistency. If one lobe is difficult to feel, it can

Box 23.25: Swellings moving on deglutition

- Thyroid swelling.
- Thyroglossal cyst.
- Pretracheal, prelaryngeal lymph node.
- Subhyoid bursa.



Fig. 23.25: Pizillo's method of thyroid examination in a patient with myxoedema



Fig. 23.26: Right thyroid lobe made prominent for examination by pushing from left side

be made more prominent by pressing firmly on the opposite side (Fig. 23.26) (**Lahey's method**).

- Feel for the position of trachea with tip of two fingers in suprasternal notch (Fig. 23.27). Normally trachea is central and it can be displaced by enlargement of one lobe of the thyroid.
- In case of tracheal compression (due to large MNG or malignant infiltration), gentle pressure on lateral lobes produces stridor (**Kocher's test**).
- Stand behind the patient and flex her neck. Using both hands, place the thumbs on nape of the neck and palpate both lobes of the thyroid with palmer surface of fingers (Fig. 23.28). While palpating, ask the patient to swallow so that nodularity is better appreciated (**Crile's method**).
- Ask the patient to swallow and see whether you can reach the lower limit of the thyroid.



Fig. 23.27: Feeling trachea in suprasternal notch



Fig. 23.30: Feeling pulsations of carotid artery (Berry's sign)



Fig. 23.28: Palpating the neck from behind the patient



Fig. 23.31: Auscultation at superior pole of thyroid for bruit



Fig. 23.29: Percussion over sternum in retrosternal goiter

- In case the lower limit of thyroid swelling is not reached, percuss over the sternum while standing in front or behind the patient and determine the extent of thyroid swelling from area of dullness (Fig. 23.29).
- Feel for the pulsations of carotid artery lateral to the thyroid (Fig. 23.30). A large benign gland displaces the carotid artery backwards and outwards so that pulsations are felt laterally. A large malignant gland surrounds the artery so that pulsations are diminished (**Berry's sign**).
- Palpate for any enlarged cervical lymph nodes that might suggest metastasis from papillary carcinoma thyroid (see Fig. 23.14).
- Auscultate at upper pole of the thyroid swelling in region of superior thyroid artery* for any systolic bruit that may be appreciated in a large vascular gland, e.g. thyrotoxicosis (Fig. 23.31).

*Superior thyroid artery is direct branch of external carotid artery and is more superficially placed.

24

Chapter

The Parathyroid and Pituitary Gland

Sanjay Marwah, Nisha Marwah

PARATHYROID GLAND

SURGICAL ANATOMY

Parathyroids are four, small, oval, yellowish brown glands arranged in two pairs. The superior parathyroids develop with the thyroid gland from 4th branchial arch. They are constant in position and are located at the termination of inferior thyroid artery on posterior surface of thyroid gland (see Fig. 23.3). The inferior parathyroids develop with thymus from 3rd branchial arch. The thymus descends into anterior mediastinum dragging with it the two inferior parathyroids. Hence, inferior parathyroids are variable in position and may be found at lower pole of thyroid, in lower part of neck or in mediastinum (within thymus) or within the thyroid substance. Blood supply of all the four glands is by inferior thyroid artery.

PHYSIOLOGY

The parathyroid cells are called chief cells that produce parathormone (PTH). The overall effect of PTH is to raise plasma calcium levels by (Fig. 24.1):

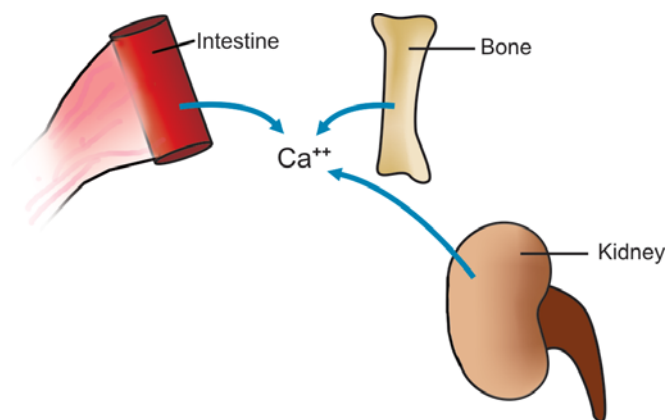


Fig. 24.1: Mechanism of rise in serum calcium by PTH

- Increasing calcium absorption from the intestine.
- Releasing calcium from bones by osteoclastic stimulation.
- Increasing tubular resorption of calcium in kidneys.

Calcitonin is the hormone secreted by parafollicular cells of thyroid gland. Its action is opposite of PTH, i.e. it lowers serum calcium levels.

HYPOPARATHYROIDISM

It is usually due to parathyroid gland damage occurring during thyroidectomy. Due to decreased PTH, there is hypocalcemia leading to tetany. In tetany, there is hyperexcitability of peripheral nerves (Box 24.1). In most of the cases it is mild and temporary. Permanent hypoparathyroidism occurs following radical thyroidectomy for carcinoma when all the four parathyroids are damaged or removed.

Clinical Features

- Initial symptoms are numbness and tingling of face (circumoral), fingers and toes (Box 24.2).
- Carpopedal spasm occurs in severe cases (Fig. 24.2). On examination of hand, thumb in palm deformity (obstetrician's hand) is seen. The fingers are extended with flexion at metacarpophalangeal joints and thumb strongly adducted.
- Laryngeal stridor may occur due to spasm of muscles of respiration leading to choking.

Box 24.1: Causes of hypocalcemic tetany

- Hypoparathyroidism.
- Chronic renal failure.
- Respiratory alkalosis.
- Metabolic alkalosis (hypokalemia).
- Vitamin D deficiency (Rickets, osteomalacia).
- Acute pancreatitis.

Box 24.2: Tetany: Clinical features

- Circumoral numbness
- Chvostek's sign
- Trousseau's sign
- Carpopedal spasm
- Laryngeal stridor



Fig. 24.2: Carpopedal spasm following thyroidectomy due to hypocalcemia

- Latent tetany can be demonstrated by following tests:
Chvostek's sign: On tapping the facial nerve in front of tragus, facial twitching occurs due to hyperexcitability of the nerve.
Trousseau's sign: On applying blood pressure cuff to the arm and inflating above systolic blood pressure, carpopedal spasm is seen in the hand.

Diagnosis

Diagnosis is by serum calcium level which is low (normal is 9-11 mg%)

Treatment

In acute cases, 10 ml of 10% calcium gluconate is given intravenous slowly over a period of 10 minutes to avoid cardiac arrhythmias. It may be repeated till the patient improves. In mild cases, oral calcium with vitamin D supplement is given.

HYPERPARATHYROIDISM

Hyperparathyroidism occurs due to increased secretion of PTH leading to hypercalcemia and its clinical manifes-

tations. Due to lack of typical presentation, high index of clinical suspicion is required to reach the diagnosis. It can be primary, secondary or tertiary.

Primary Hyperparathyroidism

It is caused by increased PTH secretion by one or more parathyroid glands. It can be due to:

- Solitary adenoma (most common –85% cases)
- Hyperplasia of all four glands (as part of multiple endocrine neoplasia syndrome)
- Parathyroid carcinoma (rare).

Secondary Hyperparathyroidism

It is a state of parathyroid overactivity induced by fall in serum calcium levels. The causes of hypocalcemia are chronic renal failure and vitamin D deficiency. The stimulus of hypocalcemia leads to hyperplasia of all the four parathyroid glands.

Tertiary Hyperparathyroidism

In case of secondary hyperparathyroidism, if parathyroid stimulus continues for a prolonged time, it can lead to formation of autonomous nodules in one or more glands. Hence, it is also known as autonomous secondary hyperplasia.

Clinical Features

Most of the cases are 'asymptomatic' and parathyroids are not palpable. The diagnosis is by hypercalcemia detected on routine biochemical screening. However, on careful examination, almost all of these patients have symptoms and can be called as 'minimally symptomatic' (Box 24.3). 'Symptomatic' cases of hyperparathyroidism are less than 50%. They have following clinical features:

- Renal stones:* Bilateral and recurrent renal stones, calcification of renal parenchyma (nephrocalcinosis).
- Disease of bones:* There is decalcification of bones leading to bone pains, formation of cysts or pseudo tumors in bones and pathological fractures. Radiological changes in form of decreased bone density and subperiosteal erosions first appear in the skull and phalanges.
- Psychic moans:* Minimal symptomatic cases in absence of serum calcium estimation are labeled as psychiatric symptoms especially in females. Such cases undergo unsuccessful treatment in mental hospitals.

Box 24.3: Clinical features of hyperparathyroidism**'Minimal symptomatic' cases**

- Muscle weakness
- Weight loss
- Constipation
- Thirst
- Headache
- Malaise
- Abdominal pain
- Depression

'Symptomatic' cases

- Renal stones
- Disease of bones
- Psychic moans
- Abdominal groans

- d. *Abdominal groans*: The patients complain of nausea, vomiting and abdominal pain. It is due to peptic ulcer and pancreatitis caused by hypercalcemia.

Diagnosis*Biochemical Investigations*

- Serum calcium levels are elevated.
- Serum PTH levels are elevated.
- Serum alkaline phosphatase levels are elevated due to bone disease.
- Serum phosphorus levels are decreased.

Radiological Investigations

- X-ray skull and phalanges show decreased bone density, subperiosteal resorption and pepper pot appearance (demineralized bone).
- Ultrasound of neck is a low cost and noninvasive investigation. It picks up adenoma in 80% cases. However, it is less sensitive in detecting ectopic lesions.
- CT scan and MRI of neck are most useful in detecting ectopic lesions in mediastinum and deep neck.
- Thallium-technetium subtraction isotope scan: Thallium outlines both thyroid and parathyroid glands while technetium outlines only thyroid gland. By subtraction of the two images with computer, all the parathyroid glands are outlined as hot spots in 95% cases.
- Selective angiography and selective venous sampling are invasive investigations and are not done

Box 24.4: Causes of hypercalcemia**Malignancy**

- Bony metastasis from primary tumors in breast, bronchus, thyroid, kidneys, prostate.
- Primary tumors producing PTH like peptides, e.g. bronchus, kidneys, ovary.

Granulomatous diseases

- Tuberculosis
- Sarcoidosis

Drugs

- Lithium
- Calcium
- Antacids

Others

- Vitamin D intoxication
- Adrenal insufficiency
- Thyrotoxicosis
- Prolonged immobilization
- Multiple myeloma.

routinely. However, in cases of recurrent hyperparathyroidism after surgery, these investigations help in localizing ectopic and missed glands.

Differential Diagnosis

It includes all the causes of hypercalcemia (Box 24.4). However, none of these conditions have raised PTH levels.

Treatment

It is surgical removal of the overactive gland or glands. The removed parathyroid should be subjected to frozen section to confirm whether it is adenoma or hyperplasia and then treated accordingly. In 90% cases there is single adenoma while remaining 10% have parathyroid hyperplasia.

1. *Adenoma*: Three glands are normal and the fourth has grossly enlarged tumor (kidney in the neck). The diseased gland is removed and biopsy taken from one normal gland. All the glands should be explored to avoid the risk of double adenoma.
2. *Parathyroid hyperplasia*: All the four parathyroids are removed and pieces of one parathyroid gland are autotransplanted in a forearm muscle. In case of recurrence, re-exploration is easy at forearm.

3. *Carcinoma parathyroid*: It is a rare condition. Radical excision including ipsilateral thyroid lobe is done taking care not to break the mass to avoid spillage.
4. *Recurrent hyperparathyroidism*: It is a difficult problem to treat. Re-exploration has high morbidity and chances of failure. Multiple endocrine neoplasia (MEN) should be excluded. Selective angiography and selective venous sampling should be done to localize the parathyroids before re-exploration.

HYPERCALCEMIA

Pathophysiology

Serum calcium is maintained by three mechanisms:

- Intestinal absorption of dietary calcium.
- Mobilization of calcium from bones.
- Renal calcium excretion

Normally 200 mg calcium/day is absorbed from the gut. Hypercalcemia occurs when normal homeostatic mechanism is disturbed.

Etiology

(See Box 24.4).

Clinical Features

These are same as seen in hyperparathyroidism. In an acute case, patient presents with severe pain abdomen, vomiting, dehydration, shock and renal failure. In untreated cases, the chances of mortality are very high.

Treatment

In acute hypercalcemia, treatment is:

- Correction of dehydration by intravenous fluids.
- Biphosphonate, in form of intravenous infusion, helps in inhibiting calcium resorption from bones.
- Calcitonin 100 IU I/M injection 12 hourly for 2 days.
- Dialysis for renal failure.

Long-term treatment:

- Maintain adequate hydration
- Oral biphosphonate
- Low calcium diet
- Steroids in sarcoidosis and RE malignancies.

PITUITARY GLAND

- The pituitary gland in an adult weighs about 500 mg and measures 13 mm × 8 mm.

- It is called **master gland** because it controls other endocrine functions.
- It is situated in the pituitary fossa (*sella turcica* of the sphenoid bone—shaped like horse saddle) in the middle cranial cavity.
- On each side of the pituitary gland lies the cavernous sinus whereas optic chiasma lies at a higher level.
- The gland consists of two lobes:

Anterior lobe (adenohypophysis)

Posterior lobe (neurohypophysis)

Anterior lobe:

- ☐ It is an ectodermal derivative formed from **Rathke's pouch**.
- ☐ It is very vascular and cellular.
- ☐ It is bigger and connected to smaller posterior lobe through a narrow zone—**Pars Intermedia**.
- ☐ Anterior pituitary is made up of **three types** of epithelial cells, each of which performs separate functions. These are:
 - *Chromophil cells with acidophilic granules*: These comprise 40% of anterior pituitary and produce:
 - i. Growth hormone (GH)
 - ii. Prolactin (PRL).
 - *Chromophil cells with basophilic granules*: These comprise 10% of anterior pituitary and produce:
 - i. Follicular stimulating hormone-Leutininising hormone (FSH-LH)
 - ii. Thyroid stimulating hormone (TSH)
 - iii. Adrenocorticotrophic hormone (ACTH), Melanocyte stimulating hormone (MSH), β lipoproteins and β endorphins.
 - *Chromophobe cells without visible granules*: These comprise remainder 50% of the anterior lobe.
- ☐ All these functions of anterior lobe are under the indirect control of hypothalamus through stimulatory and inhibitory factors which reach the anterior lobe through capillary blood flow.

Posterior lobe:

- ☐ It develops as a diverticulum from the floor of diencephalon.
- ☐ It is smaller, less vascular and made of mainly interlacing nerve fibers.
- ☐ The nerve fibers contain granules of neuro-secretory material which secrete:

- i. Vasopressin or antidiuretic hormone (ADH)
 - ii. Oxytocin
- Both of these hormones are produced by neuro-secretory cells of hypothalamus but are stored in cells of posterior pituitary.

FUNCTIONS OF PITUITARY HORMONES

Hormones of Anterior Pituitary

1. **GH:** It regulates the body growth via liver. Liver secretes somatomedin C or insulin like growth factor which helps in chondrogenesis, skeletal growth protein synthesis and cell proliferation.
2. **Prolactin:** It is active in milk production. Its secretion is high during lactation.
3. **ACTH:** It acts on adrenal cortex and regulates its secretions. If ACTH production is completely stopped, the corticosteroid secretion will also stop from the adrenal cortex leading to fatal shock (**Addisonian crisis**). Steroid replacement therapy is immediately needed as a life saving measure.
4. **TSH:** It has a regulatory effect on thyroid hormones via negative feed back mechanism.
5. **FSH and LH:** These two are known as gonadotropic hormones and control production of female sex hormones, i.e. estrogen and progesterone as well as production of testosterone.

Hormones of Posterior Pituitary

1. **ADH:** It causes re-absorption of water from the renal tubules and maintains the osmolality of plasma.
2. **Oxytocin:** It acts on myoepithelial cells of the breast leading to ejection of milk from the lactiferous ducts during lactation. It also causes contraction of uterine myometrium during delivery.

DISEASES OF PITUITARY GLAND

- Hyperpituitarism
- Hypopituitarism
- Pituitary tumors

Hyperpituitarism

- It is characterized by over secretion of one or more of the pituitary hormones.
- It may be due to diseases of anterior pituitary, posterior pituitary or hypothalamus.

- For all practical purpose, hyperfunction of anterior pituitary is due to a hormone secreting pituitary adenoma or rarely a carcinoma.

Hyperfunction of Anterior Pituitary

There are three syndromes:

- Gigantism and acromegaly
- Hyperprolactinemia
- Cushing's syndrome

- a. **Gigantism and acromegaly:** Both these syndromes occur due to sustained excess of growth hormone.

Gigantism occurs prior to closure of epiphysis in pre-pubertal boys and girls resulting in excessive and proportionate growth of child. There is both enlargement and thickening of bone with considerable increase in height and enlarged thoracic cage.

Acromegaly occurs in adults following cessation of bone growth and is more common than gigantism. The term 'acromegaly' means increased growth of extremities. There is enlargement of hands and feet, coarseness of facial features, prominent supraorbital ridges and more prominent lower jaw which when clinched results in protrusion of lower teeth in front of upper teeth (**prognathism**).

- b. **Hyperprolactinemia:** Due to excessive production of prolactin in females, it causes **amenorrhea-galactorrhea syndrome**. The latter is characterized by infertility and expression of milk from the breasts which is not related to pregnancy or puerperium. In males it may cause impotence or reduced libido.
- c. **Cushing's syndrome:** It results from ACTH excess and is mainly characterized by:
 - Central or truncal obesity with relatively thin arms and legs, buffalo hump and rounded edematous moon face.
 - Increased protein breakdown resulting in wasting and thinning of skeletal muscles, atrophy of the skin and subcutaneous tissue, osteoporosis and easy bruisability of thin skin due to minor trauma.
 - Systemic hypertension in 80% of the cases.
 - Impaired glucose tolerance and diabetes mellitus in about 20% of the cases.
 - Amenorrhea, hirsutism and infertility.
 - Insomnia, depression, confusion and psychosis.

Hyperfunction of Posterior Pituitary and Hypothalamus

These are uncommon and include two important syndromes:

- Inappropriate release of ADH
 - Precocious puberty.
- a. *Inappropriate release of ADH*: Excess release of ADH results in increased reabsorption of water and loss of sodium in the urine. It leads to expansion of intra and extracellular volume and hyponatremia. Inappropriate release of ADH occurs:
- Most often in paraneoplastic syndromes, e.g. oat cell carcinoma of lung, carcinoma pancreas, lymphoma and thymoma.
 - Infrequently due to hypothalamic lesions, e.g. trauma, hemorrhage and meningitis.
 - Rarely due to pulmonary diseases, e.g. tuberculosis, lung abscess, pneumoconiosis, empyema and pneumonia.
- b. *Precocious puberty*: It occurs due to premature release of growth hormones because of a tumor in the region of hypothalamus or pineal gland. The clinical features include:
- Premature development of genitalia
 - Growth of pubic and axillary hair
 - Breast development and onset of menstruation in females.

Hypopituitarism

- It is characterized by decreased secretion of one or more of the pituitary hormones.
- It may be due to diseases of anterior pituitary, posterior pituitary or hypothalamus.

Hypofunction of Anterior Pituitary

It occurs when there is more than 75% destruction of anterior lobe. It may result from anterior pituitary lesion or pressure and destruction from adjacent lesions. Two important syndromes are:

- Panhypopituitarism
 - Pituitary dwarfism.
- a. *Panhypopituitarism*: Three important causes are:
- Sheehan's syndrome
 - Simmond's disease
 - Empty sella syndrome.

In Sheehan's syndrome, there is pituitary insufficiency due to postpartum pituitary hemorrhage. When same process occurs without preceding pregnancy or in males, it is known as Simmond's disease.

Sheehan's syndrome is clinically characterized by:

- Failure of lactation following delivery (first manifestation)
 - Loss of axillary and pubic hair occur subsequently.
 - Amenorrhea.
 - Sterility and loss of libido.
 - There may be associated TSH and ACTH deficiency resulting in hypothyroidism and adrenocortical insufficiency.
- b. *Pituitary dwarfism*: This is due to severe deficiency of GH in children before growth is completed. Its causes are:
- Pituitary adenoma
 - Craniopharyngioma
 - Infarction and trauma to pituitary
 - Isolated inherited autosomal recessive disorder.

Clinical features appear after one year of age and include:

- Proportionate retardation in growth of bones, normal mental state for age.
- Poorly developed genitalia.
- Delayed puberty.
- Episodes of hypoglycemia.

Pituitary dwarf must be distinguished from hypothyroid dwarf (cretinism) which is characterized by mental retardation and achondroplasia.

Hypofunction of Posterior Pituitary and Hypothalamus

It is uncommon and significant clinical syndrome associated is Diabetes insipidus. Diabetes insipidus is due to deficiency of ADH and is characterized by:

- Polyuria—excretion of very large volume of dilute urine of low specific gravity (< 1010)
- Polydypsia.

Pituitary Tumors

- Pituitary tumors account for 10-15% of all intracranial tumors.
- The tumors of anterior cranial fossa are more common than those of posterior pituitary and hypothalamus.

- The majority are benign adenomas that are classified according to size, local invasiveness, patient's endocrine status, ultra structure and immunohistochemical staining.
- Carcinomas, primary or metastatic are rare. Metastases may occur, usually in elderly patients in the posterior pituitary.

Clinical Features

All pituitary tumors whether benign or malignant may present with two types of symptoms:

Pressure effects or Endocrine disturbances

1. **Pressure effects** are caused by expansion of tumors resulting in destruction of surrounding glandular tissue by pressure atrophy.

Mass effect may cause:

- Bitemporal hemianopia due to pressure on optic chiasma.
- Dysfunction of cranial nerves III, IV and VI.

Various lesions presenting as mass in sellar region are given in Box 24.5.

Box 24.5: Tumors presenting as a sellar region mass

- Pituitary tumors.
- Craniopharyngioma.
- Meningioma.
- Aneurysm.
- Rathke's cleft cyst.

2. **Endocrine dysfunction** will depend on secretory properties of the tumors if any. Secretory adenomas produce excess of corresponding clinical syndromes of hyperpituitarism. Commonly seen are:
 - Prolactinoma producing galactorrhea and primary / secondary amenorrhea.
 - Corticotroph adenoma causing Cushing's syndrome.
 - Somatotrophs adenoma causing acromegaly or gigantism.

Pituitary apoplexy is caused by hemorrhagic infarction of pituitary tumor and results in the sudden onset of headache, visual loss, ophthalmoplegia and possibly altered conscious level.

Main types of pituitary adenomas, hormones secreted by them and clinical syndromes produced are shown in Box 24.6.

Box 24.6: Pituitary adenomas

Type	Hormones produced	Clinical syndrome
Prolactinoma	PRL	Hypogonadism, galactorrhea
Corticotroph adenoma	ACTH	Cushing's syndrome
Gonadotroph adenoma	FSH-LH	Hypogonadism
Somatotroph adenoma	GH	Acromegaly/ Gigantism
Thyrotroph adenoma	TSH	Thyrotoxicosis
Nonsecretory adenoma	Nil	Pituitary failure
Pleurihormonal adenoma	Multiple hormones	Mixed

Investigations

- Eye check up
 - ☐ Testing visual acuity
 - ☐ Visual field examination
- Hormonal assay
 - ☐ Serum prolactin, FSH, LH, TSH, Growth hormone
 - ☐ Serum and urinary free cortisol
- MRI Scan of pituitary region.

Treatment

- It needs team work between neurosurgeon, endocrinologist and radiation oncologist (Box 24.7).

Box 24.7: Pituitary tumors—aims of treatment

- To alleviate mass effect
 - To restore normal endocrine function
 - To prevent recurrence
- Medical treatment:
 - ☐ Prolactinoma is treated with Bromocriptine
 - ☐ Growth hormone secreting tumor is treated with Octreotide (Somatostatin analogue) or Dopamine agonists.
 - Surgical treatment:
 - ☐ Surgery is the first line of treatment in corticotroph adenoma and thyrotroph adenoma.
 - ☐ Surgery is also indicated in Prolactinoma and Growth hormone secreting tumors that don't respond to medical treatment.

- Trans-sphenoidal surgery using operating microscope is the conventional method of surgical management. The approach is through sub-labial (underneath upper lip) or intra-nasal incision. After tumor removal, nasal packing is done for 48 hrs. The complications of surgery are given in Box 24.8.
- In recent years, endoscope is being used instead of operating microscope for tumor removal. The approach is through nostril and it causes minimal lateral damage. Nasal packing is not required after surgery and there is minimal morbidity.

Box 24.8: Complication of trans-sphenoidal surgery

- CSF leak
 - Visual deterioration
 - Major vessel injury
 - Panhypopituitarism
 - Transient diabetes insipidus
- Radiotherapy: It is given in case surgery is not possible or recurrence occurs after surgery.

25

Chapter

Swellings of the Jaw

Sanjay Marwah, Virendra Singh

The swellings arising from the jaws are classified as follows:

- I. Swellings arising from mucoperiosteum (Epulis)
- II. Swellings arising from tooth germ (Odontomes).
- III. Swellings caused by jaw tumors (Osseous and non-osseous tumors).
- IV. Inflammatory swellings.

I. EPULIS

It is a solid swelling situated on the gum arising from alveolar margin of the jaw. It can originate from mucous membrane, periosteum or bone. It has following types:

1. Fibrous Epulis

- It is the most common variety. It is a localized inflammatory hyperplasia of the gum due to irritation caused by a carious tooth.
- It forms a nodule at the junction of gum and tooth in the region of interdental papilla.
- It is a slow growing, nontender, firm polypoidal mass that often becomes pedunculated.
- Draining lymph nodes are not enlarged.
- Complications—sarcomatous change, rapid enlargement, ulceration and bleeding.
- Treatment is excision. It must be excised up to its root to prevent recurrence.

2. Granulomatous Epulis (False Epulis)

- It is a mass of granulation tissue on the gingiva situated around a carious tooth or denture.
- It looks bright red in color and feels soft or fleshy.
- It bleeds easily on touching.
- There is offensive smell in the oral cavity due to poor orodental hygiene.

- Draining lymph nodes are enlarged and tender.
- *Treatment:*
 - Maintenance of oral hygiene.
 - Removal of underlying cause (extraction of carious tooth, replacement of ill fitting denture)
 - Scraping of granulation tissue and its histopathological examination.

3. Pregnancy Epulis

There is formation of small, soft, pink mass on the gum possibly due to hormonal changes during pregnancy. The patient should improve her oral hygiene. It tends to regress after childbirth.

4. Giant Cell Epulis (Myeloid Epulis)

- It is an osteoclastoma arising from the jaw.
- Histologically, it consists of fibrocellular tissue containing multinucleated giant cells.
- It grows more rapidly than other varieties of epulis.
- It presents as hyperemic (plum colored), soft, edematous, sessile mass of the gum.
- There is underlying firm mass due to bony expansion.
- X-ray shows bone destruction (soap bubble appearance)
- *Complications:* Ulceration, hemorrhage.
- *Treatment:* Small tumors are treated by curettage and filling the cavity with cancellous bone chips. Large tumors are treated by radical excision.

5. Carcinomatous Epulis

- It is squamous cell carcinoma arising from mucous membrane of the alveolar margin.
- It presents as a non-healing ulcer that grows rapidly and fungates.

- On inner side, it invades the underlying bone.
- Regional lymph nodes are enlarged and feel hard due to metastasis.
- Diagnosis is confirmed by biopsy.
- Treatment is wide excision with a segment of bone. The defect thus created is filled with a plastic procedure.

II. ODONTOMES

These are developmental anomalies of teeth arising from epithelial or mesothelial elements. During development of the tooth, downward extension of epithelium occurs that later forms enamel organ. If a cluster of epithelial cells (epithelial debris) persists, it forms epithelial odontomes. The common varieties of **epithelial odontomes** are:

1. Dental Cyst (Radicular Cyst, Periodontal Cyst)

- It is most common of all odontomes.
- It arises from a normally erupted, chronically infected and pulpless carious tooth.
- The infection stimulates “epithelial debris” to proliferate and form a mass. This mass undergoes central necrosis, liquefaction and cyst formation.
- *Pathology*: The cyst is lined by squamous epithelium and filled with fluid containing epithelial debris and cholesterol crystals.
- *Clinical features*:
 - ☐ It is commonly seen during the middle age.
 - ☐ It is more frequently seen in the maxilla where it is mostly located anteriorly.
 - ☐ In case of dental cyst occurring in mandible, it is mostly located posteriorly.
 - ☐ It presents as a painless slow growing swelling.
 - ☐ The swelling may become fluctuant if bone is completely destroyed.
 - ☐ In later stage, it may become painful due to superadded infection (Box 25.1).
 - ☐ It may form a fistula through mucoperiosteum draining intraorally into the vestibule or extra-orally especially in the chin area.
- *Diagnosis*: It is often made on X-ray. Orthopantomogram shows a unilocular cyst, spherical or oval in shape. It is seen as a radiolucent area in relation to the root of affected tooth and its margins are sclerosed (Fig. 25.1)

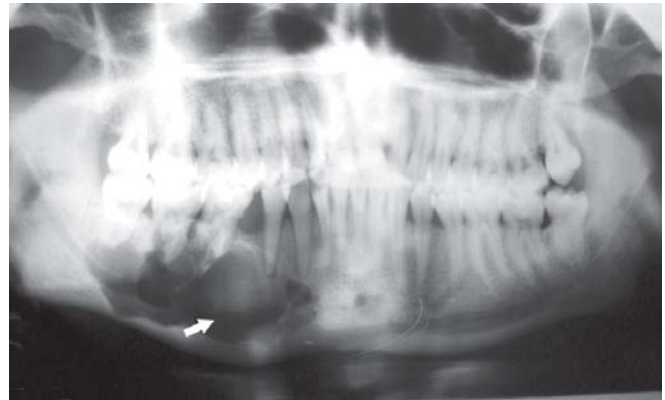


Fig. 25.1: OPG showing dental cyst of the mandible

Box 25.1: Complications of jaw cysts

- Lateral displacement of teeth
- Prevention of tooth eruption (Dentigerous cyst)
- Infection
- Sinus formation
- Pathological fracture

- *Treatment*: The affected carious tooth is removed and the cyst is excised through intraoral route. Its epithelial lining is removed, cyst wall is curetted, the cavity is filled by soft tissue ‘push-in’ and the wound is closed.

2. Dentigerous Cyst (Follicular Cyst)

- It usually occurs in relation to a non-erupted, permanent, molar tooth.
- *Etiology*: The unerupted tooth constantly irritates the epithelial cells resulting in cell degeneration and cyst formation.
- *Pathology*: The cyst is lined with squamous epithelium and filled with contents similar to dental cyst, i.e. fluid containing cholesterol crystals. The tooth lies obliquely embedded in the wall of the cyst.
- It commonly occurs in young adults and mostly involves the molars of lower jaw.
- *Clinical features*:
 - ☐ It presents as painless slow growing swelling unless secondary infection occurs.
 - ☐ It may grow very large and patient presents with progressive facial asymmetry.
 - ☐ A denture wearer may complain of alteration in the fitting of the denture.



Fig. 25.2: OPG showing dentigerous cyst of the mandible

- Clinically, a permanent tooth is missing with underlying bony expansion.
 - **Diagnosis:** X-ray shows a well-defined unilocular cyst seen as radiolucency around the crown of an unerupted tooth (Fig. 25.2). Sometimes, multilocularity is seen due to ridges of bone on the walls of the cavity.
 - **Treatment:** Total excision of the cyst through intra-oral route. The residual cavity is filled with soft tissues and bony chips.
- In case of a big cyst, it is *marsupialized*. The cyst is deroofed, contents evacuated, wall curetted and the residual cavity left open.

3. Adamantinoma (Ameloblastoma)

- It is a neoplasm of odontogenic epithelium.
- It is an epithelial tumor arising from enamel forming cells (ameloblasts).
- It is a benign slow growing tumor that behaves like basal cell carcinoma.
- It is relatively common in tropical Africa.
- **Pathology:** Grossly, it is multilocular cystic lesion filled with transparent fluid or jelly.

Microscopically, It has many variants:

- a. Follicular type—contains cuboidal or columnar cells arranged in a follicle pattern.
- b. Plexiform type—cells arranged in irregular mesh like pattern.
- c. Acanthomatous type—cells undergo squamous metaplasia with formation of keratin pearls.
- d. Granular cell type—cell cytoplasm having coarse granular appearance.

- e. Basal cell type—appearance is same as basal cell carcinoma.
- **Clinical features:**
 - It is a rare jaw tumor.
 - It is more common in mandible in molar and ramus region. It is the principal primary tumor of mandible.
 - It can rarely occur in extraoral sites as well, e.g. tibia, pituitary gland.
 - Most of the patients present in 4–5th decade.
 - It is a painless and slow growing tumor that undergoes cystic degeneration to form multiple cystic spaces.
 - It mostly causes expansion of the outer table leading to facial deformity.
 - Bony expansion and cystic degeneration may end up in a pathological fracture of mandible.
 - It feels hard at first but in advanced cases, ‘egg shell crackling’ (area of softness) can be elicited.
 - Unlike previous odontomes, it is not associated with any chronically infected or unerupted tooth.
 - **Diagnosis:** X-ray shows multiple translucent areas separated by fine bony trabeculae (Honeycomb or Soap-bubble appearance) (Fig. 25.3A).
 - **Differential diagnosis:**
 - Giant cell granuloma
 - Osteoclastoma
 - **Treatment:**
 - Since it is locally invasive tumor like basal cell carcinoma, so simple curettage or enucleation will invariably lead to recurrence. Hence, tumor should be excised with 1 cm healthy margin.
 - In case of large tumor, hemimandibulectomy may be required.
 - There is no role of radiotherapy as tumor is radioresistant (unlike basal cell carcinoma).

4. Odontogenic Keratocyst

- It arises from residual strands of epithelium from dental lamina.
- It forms a cyst in the jaw in tooth bearing area (Fig. 25.3B). The cyst is lined by keratinized squamous epithelium and has a thin fibrous capsule.
- The cyst progresses anteroposteriorly without bucco-lingual expansion of cortex.
- It contains creamy white suspension of keratin that appears like pus without any offensive smell.

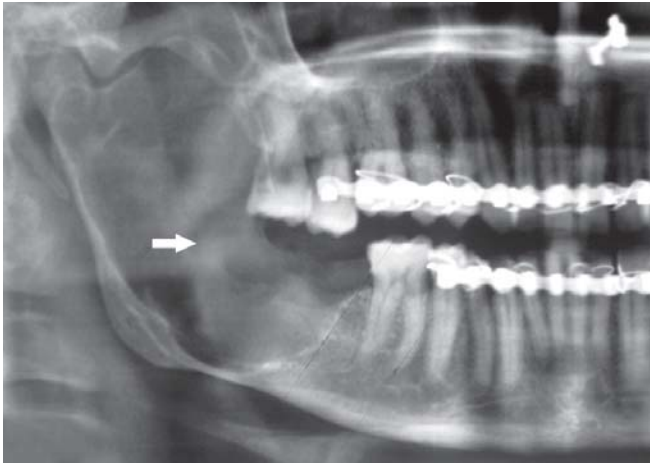


Fig. 25.3A: OPG showing multilocular radiolucency of ramus with bony expansion—ameloblastoma

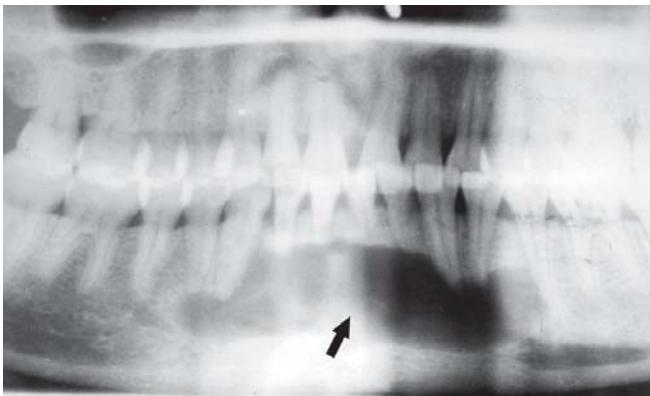


Fig. 25.3B: OPG showing unilocular radiolucency in mandibular symphysis with resorption of roots—odontogenic keratocyst

- There can be formation of multiple daughter cysts. The cyst has aggressive growth that is known to have recurrence after excision. Keratocyst is known to change to ameloblastoma or malignant lesions.
- *Treatment:*
 - ☐ Peripheral osteotomy with enucleation with chemical cautery (Cornoy's solution).
 - ☐ Resection with safe margins.

Mesothelial Odontomes

These arise from connective tissue. These are rare types and include:

- i. Fibrous odontomes
- ii. Cementomes
- iii. Sarcomatous odontomes.

There are some rare types of jaw cysts that are grouped under **non-odontogenic cysts**. These are:

i. Nasolabial Cyst

It is a developmental cyst that arises at junction of globular, lateral nasal and maxillary processes due to inclusion of epithelial cells. It presents as a swelling at the site of attachment of ala of nose. It lifts the ala of nose, forms fluctuant swelling in labial sulcus and bulges in inferior meatus of nose. It is lined by respiratory epithelium. Since it is extra-alveolar cyst, there are no radiographic findings. Treatment is complete excision through intraoral route.

ii. Nasopalatine Cyst

It is also called incisive canal cyst and is a variety of developmental cyst. It arises in incisive canal and forms a spherical bony cavity behind upper central incisors. It has a fibrous capsule and is lined by respiratory or squamous stratified epithelium. The patient may complain of pain due to pressure on nasopalatine nerve. It needs to be differentiated from dental cyst that has an associated chronically infected tooth. The treatment is surgical excision through intraoral route.

iii. Median Cyst

It is a variety of developmental cyst that produces a swelling on the palate in the midline posterior to incisive canal. X-ray shows radiolucent area with well-defined radiopaque margins. Treatment is surgical excision taking care not to damage the lining of floor of the nose.

iv. Globulomaxillary Cyst

It is a developmental cyst formed at the junction of globular and maxillary processes. It presents as a bulge between lateral incisor and canine tooth and is not associated with any non-vital tooth (cf dental cyst). The treatment is complete excision.

v. Solitary Bone Cyst (Hemorrhagic or Aneurysmal Bone Cyst)

It is believed to be traumatic in origin. Microtrauma causes intraosseous bleeding into the cancellous bone, hematoma formation and bone expansion. The cyst does not contain any epithelial lining and the wall is formed by connective tissue surrounding hemorrhagic fluid. X-ray shows unilocular or multilocular cavitation extending between teeth (Fig. 25.4). Treatment is curettage to establish fresh bleeding. The cavity is packed with gelfoam, soft tissues and bone chips followed by mucoperiosteal closure.



Fig. 25.4: Unilocular cavitation mandible—aneurysmal bone cyst

III. SWELLINGS CAUSED BY JAW TUMORS

A. Osseous Jaw Tumors

Jaws can be affected by any bone tumor. However, there are certain tumors which occur more often in the jaws. These are as follows:

1. Benign Osseous Tumors

- i. *Fibrous dysplasia:* Fibrous dysplasia is more often seen in jaw bones because these bones are membranous.

It is a benign, self-limiting but not encapsulated and diffuse lesion of the bone. The normal bone is replaced by fibrous tissue containing islands of metaplastic bone. It occurs during growing age leading to deformity of the jaws with disturbance in eruption pattern of teeth. The bony enlargement usually stops once skeletal growth is complete and surgery should be deferred till this stage. Surgery during growing age may result in recurrence. The fibrous dysplasia can affect the jaw bones in two forms:

Monostotic lesion: Single bone is involved usually affecting maxilla (Fig. 25.5A).

Polyostotic lesion: Multiple bones are involved. Skull and jaws are almost always involved. It may occur as part of Albright's syndrome (Box 25.2)

In fibrous dysplasia, X-ray picture shows rarefied areas in the medullary portion of the bone with irregular trabeculations giving multilocular cystic appearance (*Cotton-wool appearance*). CT scan shows bony expansion with mixed radiolucency (Fig. 25.5B).



Fig. 25.5A: Swelling of left side face obliterating nasolabial fold—fibrous dysplasia

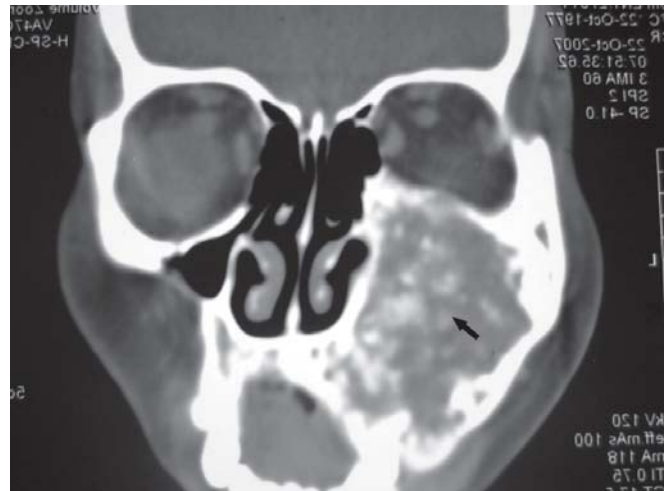


Fig. 25.5B: CT scan (coronal section) showing mixed radiolucency of left maxilla displacing orbital floor—monostotic fibrous dysplasia

Box 25.2: Albright's syndrome

- Polyostotic fibrous dysplasia
- Cutaneous pigmentation
- Endocrine disorders (Diabetes)
- Precocious puberty
- Premature skeletal maturation

- ii. *Ossifying fibroma:* It is a benign neoplasm containing fibrous tissue with islands of metaplastic bone like fibrous dysplasia. However, unlike fibrous dysplasia, it is circumscribed and

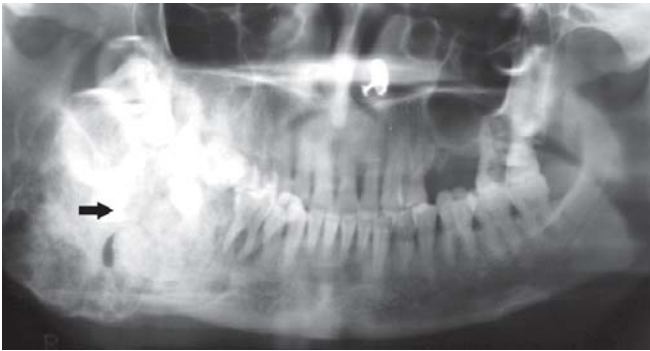


Fig. 25.6: OPG showing ossifying fibroma of mandible

capsulated lesion. It is generally seen in adults and involves both the jaws. Its consistency is variable depending upon the degree of calcification. X-ray shows mixed radiopaque and radiolucent lesion with more radiodensity in center than at periphery. It causes bony expansion and tooth displacement (Fig. 25.6).

- iii. *Paget's disease of the jaw:* It may arise as a part of generalized Paget's disease or may rarely be confined to the jaws. It mostly affects the maxilla and the lesion progresses through the face into the vault of the skull. There is involvement of bone and teeth. There is osteoclastic resorption followed by osteoblastic regeneration of the bone that is highly vascular. The affected bone gradually enlarges and becomes dense and sclerotic. Teeth exhibit hypercementosis in a mosaic pattern. Ankylosis of teeth is also commonly seen. The patient presents with facial deformity and difficulty in wearing of dentures due to enlargement of ridges. Sometimes the patient presents with inability to wear his normal size of hat due to progressive thickening of bones of the vault. There can be brisk hemorrhage following tooth extraction due to increased bone vascularity.

Investigations: Serum alkaline phosphatase levels are very high.

X-ray appearance: The involved bone is bigger than normal, cortex is thickened and medullary cavity shows patchy sclerosis.

Complications: It may rarely change to osteosarcoma.

- iv. *Osteoclastoma:* It is extremely rare tumor of the jaw mostly affecting lower jaw. It usually affects young males. There is rapid growth and both the

tables of bone are thinned out leading to substantial destruction of the jaw. X-ray shows radiolucent cysts (*Soap-bubble appearance*) and ill-defined trabeculae (*pseudotrabeculae*). Treatment is wide excision.

- v. *Giant cell granuloma:* It is also known as giant cell reparative granuloma. Its etiology is unknown. It may be traumatic in origin leading to hemorrhage within the bone marrow.

Pathology: Grossly, it consists of opaque, dark red, semisolid material.

Microscopically, it has unevenly distributed multinucleated giant cells, connective tissue cells, collagen and thick walled blood vessels.

Clinical features: It commonly affects young females (10-25 years). It presents as a lobulated mass in the central part of the jaw, usually mandible (Fig. 25.7A). The lesion is painless and grows by expansion and thinning of cortical plates.

X-ray: It shows rounded or oval translucent area that expands the cortex with subperiosteal new bone formation.

CT scan: It shows circumscribed bony expansion that is translucent (Fig. 25.7B).

Differential diagnosis (see Box 25.3):

- (a) Adamantinoma
- (b) Osteoclastoma
- (c) *Brown tumor of hyperparathyroidism:* Serum calcium and serum parathormone levels are high. X-rays of other parts of skeleton (distal phalanges) show patchy osteolytic lesions.

Treatment:

- Enucleation with primary closure of mucosal flap.
 - Gentle but thorough curettage of the cancellous bone in the wall of the cavity.
- vi. *Cherubism:* It manifests in early childhood (2-4 years) and tends to regress after puberty. There is painless, progressive, symmetrical swelling of the jaws producing a chubby face, hence, named "Cherubism". It commonly affects the mandible. Extensive lesions interfere with tooth development and eruption and even erupted teeth may be lost early. Regional lymphadenopathy may be present. X-ray shows extensive bilateral destruction of bone with expansion and severe thinning of cortical plates.



Fig. 25.7A: Peripheral giant cell granuloma located on lingual aspect of dentoalveolar ridge



Fig. 25.7B: CT film (axial section) showing circumscribed translucent expansion of maxilla obliterating nasal cavity—giant cell granuloma

Box 25.3: Differential diagnosis of giant cells granuloma

	<i>Giant cell granuloma</i>	<i>Adamantinoma</i>	<i>Osteoclastoma</i>
Incidence	Young females (10-25 years)	Males 40-45 years	Males, 25-40 years
Etiology	Traumatic	True epithelial neoplasm of ameloblasts	Tumor of giant cells
Progress	Slow growth	Slow growth, locally invasive	Rapid growth
Site	Mandible (central part)	Mandible (molar and ramus)	Mandible
Bony expansion	Both inner and outer tables	Outer table	Both inner and outer tables
Fungation	No fungation	May fungate outside in later stages	No fungation
X-ray picture	Round to oval translucent area that expands cortex with subperiosteal new bone formation	Multiple translucent areas separated by fine bony trabeculae (Honeycomb appearance)	Soap bubble appearance with ill-defined trabeculae (pseudotrabeculae)
Microscopy	Unevenly distributed multinucleated giant cells, few in number	Many variant ranging from follicular type to basal cell type	Large number of multinucleated giant cells in fibrocellular stroma.
Treatment	Enucleation/curettage	Wide excision with safe margin	Wide excision
Recurrence	Does not recur	It recurs commonly	It recurs commonly
Radiosensitivity	RT not indicated (benign lesion)	Radioresistant	Radiosensitive

Microscopically: It has multinucleated giant cells, whorled fibrous tissue, thin walled blood vessels and hemosiderin (resembles giant cell granuloma). Enlarged lymph nodes contain hemosiderin.

- vii. *Osteoma:* It is slow growing and is composed of mature bone. It is seen most frequently in the

mandible. It can be *central (endosteal)* or *peripheral*.

- a. *Central osteoma:* It is an outgrowth from inner surface of cortex and causes localized expansion of the jaw. X-ray shows a well-demarcated radiopaque area in the bone that is surrounded by a radiolucent line.



Fig. 25.8: Operative photograph showing peripheral osteoma being excised

- b. *Peripheral osteoma*: It arises from periosteum of underlying bone and presents as pedunculated rounded lump on the surface of the jaw (Fig. 25.8). Surface is extremely hard. The patient presents with cosmetic deformity and pain. Multiple osteomas of the mandible may be seen as a part of Gardner's syndrome (Box 25.4).

Box 25.4: Gardner's syndrome

- Multiple osteomas of mandible
- Multiple osteomas of frontal bone
- Multiple polyps of the colon
- Leiomyoma of the stomach
- Desmoid tumors in surgical scars
- Compound odontomes
- Impacted supernumerary and permanent teeth

On X-ray: Bone in the area of tumor is densely sclerotic.

Treatment: It can be ignored if it is asymptomatic. If it is painful or causing cosmetic deformity, treatment is surgical excision.

- viii. *Chondroma*: It is slow growing tumor arising from mature cartilage or from precartilaginous connective tissue. Maxillary chondromas are more common. It can occur at any age and has no sex predilection. It forms nontender, painless, sessile swelling that can attain an enormous size. It can undergo a malignant change (chondrosarcoma).

Treatment: It should be surgically excised with 1 cm tumor free margin because it is difficult to differentiate chondroma from chondrosarcoma even histologically. Moreover, it is radioresistant.

2. Malignant Osseous Tumors

The malignant osseous tumors differ from benign osseous tumors (Box 25.5).

Box 25.5: Differences between benign and malignant osseous jaw tumors

Benign tumors	Malignant tumors
• Slow growing • Painless (except osteoid osteoma) • No systemic signs and symptoms • Don't cause root resorption • No anesthesia, paresthesia • Overlying mucous membrane remains intact • X-ray—lesion is solitary and well-circumscribed (except fibrous dysplasia) • Treatment—local excision	• Rapid growing • Painful • Systemic signs and symptoms present • Root resorption and tooth mobility seen • Anesthesia and paresthesia due to nerve involvement • Ulceration and fungation of mucous membrane • X-ray—extensive lesion with ill defined edges • Treatment—wide excision

Following are the malignant osseous tumors:

- i. *Osteogenic sarcoma*: It is an uncommon but highly malignant tumor of the jaw. It occurs in children during period of active growth (10-30 years). Mandible is more commonly affected than maxilla. *Clinical features* are pain, rapidly progressive jaw swelling, loosening and displacement of teeth. X-ray shows typical "Sun ray appearance" due to radiating spicules of bone extending outward from the cortex (Fig. 25.9). *Treatment* is radiotherapy followed by radical surgery. *Prognosis* is better than osteogenic sarcoma of long bones. Five years survival is 25-35%.
- ii. *Chondrosarcoma*: It is also a rare jaw tumor. It is difficult to differentiate from chondroma even on histopathology.

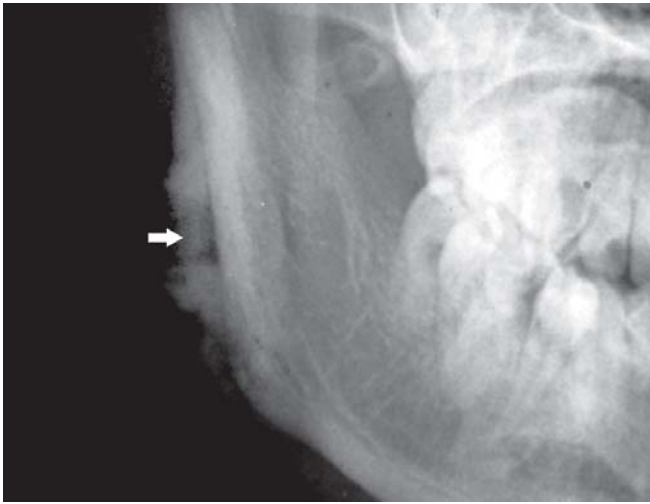


Fig. 25.9: PA view mandible showing 'sun ray' appearance with periosteal elevation—osteogenic sarcoma

Clinical features suggestive of malignancy are appearance of pain, rapidly growing tumor, displacement and exfoliation of teeth.

Treatment is radical surgical excision.

B. Non-osseous Jaw Tumors

These can be benign or malignant tumors. These are classified based on their tissue of origin (Box 25.6).

Many of these lesions have been described in Chapter 11: Tumors.

1. Benign Non-osseous Tumors

These usually present as central tumors of the jaws. Clinical features and radiographic appearance is not able to give definitive preoperative diagnosis in most cases. A biopsy is the only certain means of preoperative diagnosis. However, in a vascular lesion, open biopsy is contraindicated and may even prove fatal. Therefore, in a suspected vascular lesion, before performing open biopsy, aspiration should be attempted first through intact mucosa or intact bone to know the nature of the lesion.

Treatment is excision of the tumor in symptomatic cases.

2. Malignant Non-osseous Tumors

Squamous cell carcinoma is the commonest malignant tumor affecting the jaws.

Box 25.6: Non-osseous jaw tumors

Tissue of origin	Benign	Malignant
Surface epithelium	Papilloma	Squamous cell carcinoma
Basal cell layer of skin	—	Basal cell carcinoma
Neuroectoderm	Benign melanoma	Malignant melanoma
Glandular epithelium	Adenoma	Adenocarcinoma
Adipose tissue	Lipoma	Liposarcoma
Fibrous tissue	Fibroma	Fibrosarcoma
Smooth muscles	Leiomyoma	Leiomyosarcoma
Skeletal muscles	Rhabdomyoma	Rhabdomyosarcoma
Nerve cells	Neurofibroma	Neurofibrosarcoma
Lymph vessels	Lymphangioma	Lymphangiosarcoma
Blood vessels	Hemangioma	Angiosarcoma
Hemopoietic cells	—	Leukemia/Multiple myeloma
Marrow epithelium	—	Ewing's sarcoma
Salivary glands	Pleomorphic adenoma	Malignant pleomorphic adenoma
Secondary deposits	—	Metastatic tumor (thyroid, lungs, breast, neuroblastoma)

i. Malignant tumors of the mandible

- Mandible hardly ever has primary malignant neoplasm and is mostly involved from advanced cancer of adjoining structures (tongue, floor of mouth, cheek) that is usually **squamous cell carcinoma**.
- The tumor invades the underlying bone quite early. It must be assumed that spread along inferior dental canal has occurred and the bone from proximal part of mandibular foramen to beyond the mental foramen should be excised.
- If mandible has superficial invasion by tumor spreading from adjoining tissues, **en bloc excision** can be done while conserving the opposite cortex. It helps in maintaining continuity of the jaw.
- In larger tumors, **segmental resection of mandible** or **hemimandibulectomy** is done including generous removal of adjoining soft tissues.

- If cervical nodes are involved, **block dissection of neck** is also done.
- If resection of mandible is short of midline, it is not essential to replace the mandible and the defect is filled with appropriate flaps (Pectoralis major myocutaneous flap) for acceptable function and appearance. Primary reconstruction at the time of surgery is always better than delayed reconstruction. If mental region of the mandible is also excised, mandibular replacement (by cancellous bone from iliac crest, rib or titanium implant) is essential to give stability and to permit suspension of the larynx.
- These days **microvascular free flaps** containing soft tissues and vascularized bone are being used that give very good cosmetic as well as functional results. The arteries and veins of free flap are anastomosed with the arteries and veins of local tissues. The examples are radial forearm flap containing a segment of radius bone, free fibula flap and compound groin flap containing segment of iliac crest.
- **Radiotherapy:** Orthovoltage radiotherapy has risk of radionecrosis of the jaw. Megavoltage therapy is more effective and has lower incidence of bone necrosis. Radiotherapy is combined with surgery in large tumors.
- Other malignant tumors involving the mandible are:
 - ☐ Metastatic deposits
 - ☐ Lymphoma
 - ☐ Multiple myeloma
 - ☐ Histiocytosis X.

Metastatic Deposits

Although rare in mandible, metastases are the most common bone tumors in adults. Carcinomas having bony metastases are mostly those that arise from paired midline organs, i.e. breast, lungs, prostate, kidneys, adrenals, thyroid. They usually present with local pain and pathological fracture in a patient with past history of cancer. Metastases are almost always multiple and are best demonstrated on bone scan. X-ray shows multiple osteolytic areas and pathological fracture.

Treatment: Apart from treating primary lesion, treatment for metastasis is palliative in form of radiotherapy and surgery (internal fixation for stabilization of pathological fracture).

Multiple Myeloma

Although rarely seen in mandible, it is the most common primary malignancy affecting bones in adults. There is multiple or diffuse bone involvement. It is believed to originate from bone marrow cells. It is mostly seen in people between 4-7th decade of life. Patient mostly complains of the back pain.

Common sites of involvement in the mandible are ramus, angle and molar region. The lesion destroys the bone replacing it with soft fleshy purplish tissue. Pathological fracture may occur.

Investigations:

- ESR is raised.
- Elevated serum and urine Bence Jones proteins.
- Monoclonal gammopathy.
- X-ray shows multiple lytic, sharply punched out lesions.
- Bone scan shows no evidence of bone formation (cold spots).
- Biopsy shows dense mass of cells resembling plasma cells.

Treatment: Cytotoxic chemotherapy and local excision. Radiotherapy is given to decrease tumor size.

Histiocytosis X

The histiocytic cells serve as a number of defensive and physiological functions in the body. These are wide spread in organs but are heavily concentrated in spleen, liver, lymph nodes, bone marrow and blood.

Histiocytosis X is a group of lesions derived from Langerhans cells (histiocytic cells). Oral signs and symptoms of bone and soft tissue involvement may be initial clinical manifestations. Histiocytosis exhibits a spectrum of clinical expressions:

- Eosinophilic granuloma:** It is the mildest expression of histiocytosis X and is usually confined to one bone. In oral cavity, there is swelling and pain over the jaw lesion. The gum becomes ulcerated, red granulation tissue appears and secondary infection occurs. The teeth in involved area become mobile and healing does not occur after tooth extraction.
- Hand-Schuller-Christian disease:** It is a systemic extension of eosinophilic granuloma. It usually occurs in children and young adults. The systemic features are:
 - ☐ Anemia

- ☐ Weight loss
- ☐ Growth disturbance
- ☐ Neural dysfunction
- ☐ Hepatosplenomegaly
- ☐ Lymphadenopathy

Bone lesions are found in skull, jaws, ribs, pelvis and long bones. X-ray skull shows extensive punched out areas of destruction.

- c. *Letterer-Siwe disease*: It is an acute disseminated form of histiocytosis X. It primarily occurs in infants during first year and in young children (below 3 years). All signs and symptoms become acute and widespread. Oral lesions may show severe inflammatory hyperplasia, ulceration and necrosis of gums and other soft tissues. X-ray picture resembles osteomyelitis in appearance.

Histopathology shows proliferation of histiocytic cells, large number of eosinophils along with few lymphocytes, plasma cells and neutrophils.

Treatment of histiocytosis X is based on extent of clinical involvement. A single lesion of the jaw is treated with curettage. In disseminated histiocytosis X, curettage of bone lesion is combined with systemic steroids and chemotherapy (alkaloids, vinblastine).

Prognosis depends upon the clinical course. A rapid onset systemic involvement without bone lesions has grave prognosis. Single or multiple bone lesions offer a favorable prognosis.

ii. Malignant Tumors of the Maxilla

Carcinoma maxillary antrum is of two varieties:

- a. *Squamous cell carcinoma*: It arises from epithelium lining the hard palate or gum. It is more common in India due to betel chewing, smoking (reverse smoking).
- b. *Adenocarcinoma*: It is columnar cell carcinoma maxillary antrum. It may occur as occupational disease in wood workers, chromic and nickel industries.

Clinical features:

- It is mostly seen after 40 years of age.
- Initially, it is symptomless.
- Obstruction of ostium and infection of secretions give symptoms like chronic sinusitis.

- Growth on floor of the antrum presents with dental symptoms due to early alveolar invasion. The patient presents with pain, bulge of hard palate, loose teeth, ill-fitting dentures, oroantral fistula.
- Growth on medial wall presents with nasal obstruction, blood stained purulent discharge. There is epiphora due to obstruction of nasolacrimal duct.
- Growth involving anterolateral wall presents with pain in the cheek. There is anesthesia of the cheek, anterior teeth and gums due to involvement of infraorbital nerve. There is obvious bulging on the cheek due to growth that may ulcerate and fungate.
- Growth spreading superiorly presents with diplopia and proptosis due to invasion of orbital floor.
- Growth spreading posteriorly may not give significant symptoms until invasion of base of skull has occurred. There is trismus due to involvement of pterygoid muscles. There is paresthesia over cheek, gums, lower lip and tongue due to nerve involvement. There is post-nasal blood stained discharge due to ulceration of growth into nasopharynx. It carries poor prognosis due to late presentation.
- Lymph node metastasis occurs late and involves upper deep cervical nodes. It carries poor prognosis.

Diagnosis:

- Biopsy by Caldwell Luc operation (intranasal antrostomy) confirms the diagnosis.
- CT scan helps in defining exact extent of the lesion.

Treatment:

- Radiotherapy is main mode of treatment. Mega-voltage radiotherapy is given for six weeks. It is curative in about 70% cases. In advanced cases, radiotherapy reduces tumor bulk and makes it resectable.
- Surgery is done in early localized tumor or in case of residual disease after radiotherapy. Total maxillectomy is done if growth involves entire maxilla.
- A tumor confined to hard palate, upper alveolus and floor of the antrum can be resected by conventional partial maxillectomy. The resultant cavity after maxillectomy should be skin grafted to ensure rapid healing and to prevent contracture of soft tissues.
- The defect created after surgery requires prosthesis or reconstruction for cosmesis. Reconstruction can be done by temporalis muscle flap or microvascular flap.

- Metastatic deposits in cervical lymph nodes require block dissection of the neck.
- Chemotherapy can be given if recurrence occurs after surgery and radiotherapy.

Other less common malignant tumors of maxilla are:

- Malignant tumor of minor salivary glands.
- Malignant melanoma (See Chapter 11: Tumors)
- Burkitt's lymphoma (See Chapter 13: Diseases of Lymph Nodes)
- Ewing's sarcoma: It is uncommon malignant neoplasm that arises from endothelial lining of blood vessels. It occurs mostly during first two decades of life. It presents as a primary destructive lesion of the bone. The symptoms are pain, fever, jaw swelling and interference with jaw functions. X-ray shows 'onion peel' appearance due to subperiosteal new bone formation over areas of bone destruction. The most characteristic feature of Ewing's sarcoma is enormous extraosseous soft tissue component that is far more than area of bone destruction. Treatment is radiotherapy to involved area and chemotherapy.

IV. INFLAMMATORY SWELLINGS

Pericoronitis

Once an erupted tooth has penetrated overlying soft tissues, a potential cleft remains between enamel surface and adjacent tissues. It is a potential site for

infection and such infection is virtually confined to lower third molar tooth. The patient presents with soreness and pain in lower third molar region, redness and edema of the gum, swelling of the cheek, trismus and tender submandibular lymph nodes. If untreated this infection can progress further producing alveolar abscess (Box 25.7).

Treatment

- During pericoronitis, treatment is antibiotics that cover both aerobic and anaerobic bacteria (amoxycillin + metronidazole), anti-inflammatory drugs and mouthwashes.
- Once pericoronal abscess forms, treatment is intra-oral incision and drainage under cover of antibiotics.
- Third molar tooth should be extracted if possible at the time of drainage after acute symptoms subside.

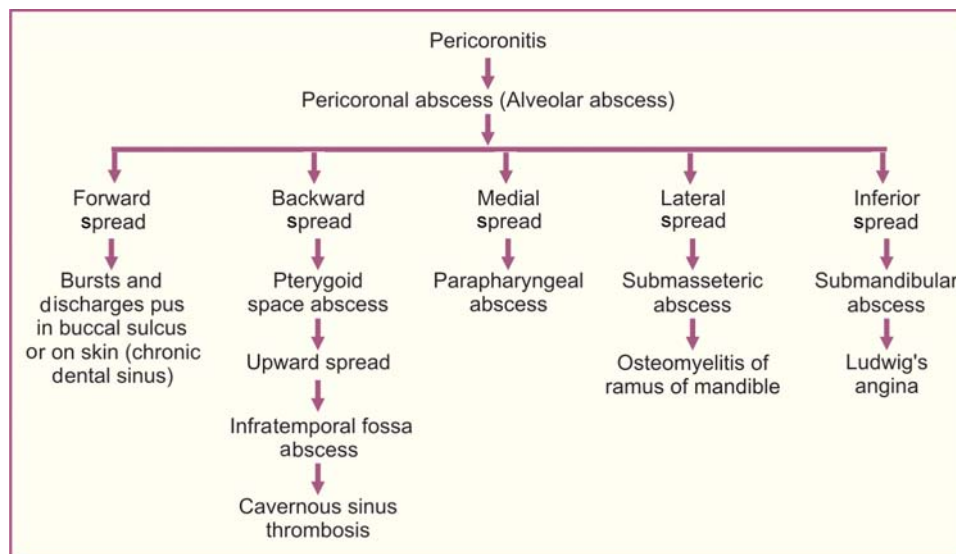
Complications of Alveolar Abscess

- Osteomyelitis of jaw
- Ludwig's angina (See Chapter 3: Infections)
- Cavernous sinus thrombosis
- Chronic dental sinus

Chronic Dental Sinus

When the alveolar abscess bursts on the skin, it results in formation of a non-healing sinus. The sinus opening

Box 25.7: Mode of spread of infection in pericoronitis



may or may not overlie the affected tooth as it always follows the path of least resistance which is further determined by periosteum and direction of muscle fibers. Pus from the lower incisors can penetrate buccal cortical plate below the origin of mentalis muscle. The pus reaches the surface between two muscles and drains via a sinus in the middle of the chin and named as *Median mental sinus* (see Fig. 5.3).

Clinical Features

- The patient complains of painless, chronic, non-healing sinus.
- Its typical location and appearance makes it a spot diagnosis.
- On palpation, a sinus track can be felt as a fibrous cord beneath sinus opening that leads to the underlying thickened bone.
- Examination of oral cavity reveals evidence of infected tooth (odontogenic infection).
- If clinician is unaware of this condition, it is invariably diagnosed as infected sebaceous cyst.
- Misdiagnosis leads to local excision that is always followed by recurrence.

X-ray mandible may show periapical bone destruction or may reveal nothing abnormal. Intraoral periapical X-ray shows widening of periodontal space and area of rarefaction around the root.

Treatment

Extraction of infected tooth leads to spontaneous healing of the sinus. If there is chronic ugly facial skin scar, it should not be excised for at least six months after tooth extraction since it may improve with time. If it persists even after that, it is excised by transverse elliptical incision (along lines of Langer). The sinus track is curetted and wound is closed with fine sutures.

Osteomyelitis of the Jaws

It is extensive inflammation of the bone including marrow spaces, cancellous bone, cortex and periosteum. In adults, it is more common in mandible while in infants; it is more common in maxilla.

Classification

- Acute osteomyelitis
- Chronic osteomyelitis

- Chronic osteomyelitis associated with specific infection
 - ☐ Tuberculosis
 - ☐ Actinomycosis
 - ☐ Syphilis
- Necrosis of the jaw
 - ☐ Radiations
 - ☐ Chemicals
 - ☐ Electrocoagulation

i. Acute Osteomyelitis

- In infants:* It often occurs in infants involving maxilla.

Etiology

- Birth trauma causing abrasion of palatal mucosa.
- Infection through feeding bottles or unclean nipple of the mother.
- Hematogenous infection by streptococci or pneumococci.

Clinical features: The baby is severely ill with high grade fever, vomiting and delirium. The first sign is appearance of redness and swelling below the inner canthus. The upper and lower eyelids become puffy and there is discharge of pus from the nostril on affected side. Abscess forms on alveolar margin and bursts to discharge pus. Fever comes down once there is pus discharge.

X-ray findings are inconclusive during early stage.

Treatment

- Parenteral antibiotic cover, intravenous fluids.
- Pus culture and sensitivity to guide the antibiotic treatment.
- Intraoral drainage of abscess.
- Later, sequestrectomy may be required.

- In children and adults:*

Etiology:

- Spread of alveolar abscess
- Infection of cysts and tumors
- Fracture of the jaw
- Maxillary sinusitis
- Tonsillitis
- The causative organism is usually *Staph aureus*.

Pathogenesis: It mostly affects mandible due to presence of single tenuous blood supply along its long axis that is easily obstructed by infection or trauma. It leads to ischemic necrosis with superadded bacterial infection

leading to osteomyelitis. The pus gets collected under the periosteum that gets raised from the underlying bone interrupting the periosteal vessels. A line of separation appears between necrosed and healthy bone. Necrosed bone finally gets separated and is known as **sequestrum**. Surrounding sequestrum, elevated periosteum lays down new bone that ensheaths the dead bone. This new bone is called **involucrum**. The pus discharges through small perforations in the involucrum and these holes are known as **cloacae**. Such advanced pathological changes are rarely seen these days because of modern antibiotics.

Clinical features:

- Gradually increasing pain.
- High grade fever with chills.
- Offensive halitosis.
- Affected area is tender on palpation.
- Involved teeth are loosened.
- Swelling and unilateral numbness of lip (due to involvement of inferior dental nerve).
- Gum mucosa is angry looking and inflamed.
- Cellulitis of face.
- Trismus due to involvement of muscles of mastication.
- In extensive lesion, pathological fracture may occur.
- In osteomyelitis of maxilla there are ocular symptoms in form of epiphora, proptosis, impaired eye movements and even blindness.

X-ray:

- No significant findings during initial period.
- After 10 days X-ray may show:
 - ☐ Multiple small radiolucent patches.
 - ☐ Moth eaten appearance due to scattered areas of bone destruction.

Treatment:

- **Medical management:** Antibiotics, analgesics, intravenous fluids for hydration, bed rest and high protein diet.
- **Surgical management:** Incision and drainage of pus under cover of antibiotics. A small soft rubber drain is inserted in the cavity to facilitate drainage. Cavity is irrigated with saline and regular dressing is done. The offending tooth is extracted.

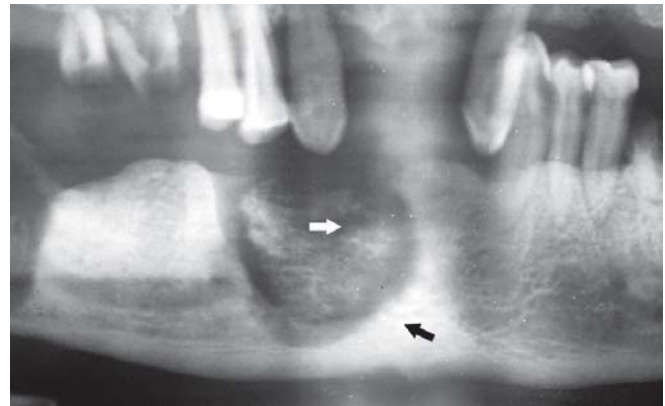


Fig. 25.10: OPG showing bony destruction of mandibular body containing sequestrum surrounded by involucrum

ii. Chronic Osteomyelitis

It may occur secondary to acute osteomyelitis or following primary infection by organisms of low virulence.

Clinical features:

- Mandible is affected more commonly than maxilla.
- Patient is not toxic.
- Constitutional symptoms are less severe.
- There is dull aching pain in the jaw.
- There is chronic discharging sinus in oral cavity or on the skin.
- On palpation underlying bone is thickened and mildly tender.
- Sometimes dead bone pieces (sequestrum) are discharged through the sinuses.

X-ray findings: It appears after 3 weeks of infection. X-ray shows radiopaque mass in the center (sequestrum) surrounded by radiolucent line (Fig. 25.10). The sequestrum appears whiter than surrounding bone due to its high calcium contents. Periosteum is separated from the cortex and seen as white line emerging from the cortex.

Treatment: It is primarily surgical treatment. Sequestrectomy and saucerization is done. A suitable incision is made at the dependent part of the affected area of mandible. The involucrum is chiseled and the cavity is made open (saucerization). The sequestrum in the cavity is removed and the residual granulation tissue is curetted till white shiny bone appears. Bleeding is controlled by pressure packs. A glove drain is placed in the cavity and changed every 24 hours till discharge ceases from the cavity. Appropriate antibiotics are given.

Complications during surgery:

- Bleeding
- Injury to inferior dental nerve
- Pathological fracture.

iii. Chronic Osteomyelitis Associated with Specific Infection

a. *Tubercular osteomyelitis of jaw:* It is uncommon disease and is mostly seen in young children. It is always associated with tubercular infection elsewhere.

Mode of spread:

- Direct extension from gingival lesion.
- Direct extension from infected sputum through extraction socket.
- Hematogenous spread.
- Local spread from tubercular submandibular lymph node.

Clinical features:

- Painless swelling of the jaw.
- Mildly tender on palpation.
- Loosening of teeth.
- Formation of pus discharging sinuses.
- Enlargement of regional lymph nodes that may show matting and caseation.

X-ray: shows features of chronic osteomyelitis.

Treatment:

- Antitubercular drugs.
- In case of persistent sinuses, treatment is electro-cauterization.
- Extensive jaw involvement may require jaw resection.

b. *Actinomycosis of the jaw:* See Chapter 4: Specific Infections.

c. *Syphilis of the jaw:* It is rarely seen these days. The bones are affected in tertiary stage. Commonly affected areas are cranial bones, nasal bones and hard palate. Bone lesions result from formation of 'gumma' (See Chapter 4: Specific Infections). Mandible is usually affected and features are similar to pyogenic osteomyelitis. Treatment is same as for pyogenic osteomyelitis.

iv. Necrosis of the Jaw

a. *Radiation necrosis:* It occurs as a complication of radiotherapy especially deep radiation therapy. It is also

seen in radium dial painters who lick their brushes during painting. Massive dose of radiation causes bone necrosis due to thrombosis of blood vessels. Secondly infection supervenes leading to radiation osteomyelitis.

Clinical features:

- Deep boring pain in the jaws.
- Ulceration of mucosa and skin in overlying area that fails to heal.
- Once infection occurs, there is jaw swelling along with trismus.
- There is abscess formation that bursts to form multiple discharging sinuses.

Treatment:

Prophylactic

- Before giving radiotherapy to jaws, all foci of infection should be removed from the jaws (e.g. infected teeth).
- Fluoride therapy to prevent radiation caries.
- Avoid tooth extraction in heavily irradiated jaws.

Medical

- Local and systemic antibiotics.
- Good oral hygiene.
- Hyperbaric oxygen.

Surgical

- Sequestrectomy and resection of the jaw.

b. *Chemical necrosis:* It is rarely seen today. The chemicals causing necrosis of jaws are phosphorus, arsenic and mercury.

In *phosphorus necrosis*, involucrum completely surrounds the sequestrum and large window is required to be made to remove the underlying sequestrum. It is seen in match factory workers.

Mercury poisoning occurs from its medicinal use. The bone becomes denuded and osteoradionecrosis occurs.

c. *Osteonecrosis due to electrocoagulation:* Electrocoagulation is widely used in oncosurgery. When used adjacent to bone, it can cause bone necrosis. Its heat kills soft tissues and periosteum exposing underlying bone to infection. However, early sequestration occurs that gets separated and continuity of jaw is maintained.

Treatment:

- Oral prophylaxis.
- Obtundent dressing.
- Antibiotics to prevent secondary infection.

CLINICAL EXAMINATION OF THE JAWS

Examination of Maxilla

- Maxilla has five surfaces for examination:
 - ☐ *Superior surface (orbital surface)*: It forms floor of the orbit. Compare inferior orbital margins on two sides by palpation and note any difference. Upward bulging of orbital floor can push eye ball forwards (proptosis).
 - ☐ *Superficial surface*: There can be excess flow of tears (epiphora) on face due to blockage of nasolacrimal duct by a maxillary tumor. Look for any bulge in the cheek and feel it after evertting upper lip.
 - ☐ *Inferior surface (palatine surface)*: Ask the patient to open mouth and examine the palate for any swelling. Also examine the teeth in upper jaw.
 - ☐ *Medial surface (nasal surface)*: Ask the patient to blow nose by occluding nares one at a time to check the patency. In case of unilateral nasal obstruction, examine the affected side with nasal speculum.
 - ☐ *Posterior surface*: This surface is difficult to examine since it is beyond our reach. However, growth from this surface extends to involve infratemporal region and then temporal fossa. So temporal region should be palpated for any fullness.
- Tenderness of maxillary antrum without any mass and associated unilateral purulent nasal discharge in an infant suggests acute osteomyelitis.
- Solid mass arising from mucoperiosteum is epulis. Note its size, base (sessile/pedunculated), surface (smooth/ulcerated), consistency (soft/firm), bleeds on touching or not.

- Do transillumination test for maxillary antrum.
- Examine cervical lymph nodes for enlargement.
- Examine maxillary division of trigeminal nerve (involved in malignancy).

Examination of the Mandible

- Look for any obvious deformity, swelling or sinus in the region of lower jaw.
- The palpation of mandible is done bimanually by keeping one finger within the mouth and the fingers of other hand applied externally.
- The body, angle and inferior part of ramus are easily palpated while superior part of ramus and condyloid and coronoid processes are palpated with difficulty.
- In case of any swelling, note its site, size, shape, surface, consistency, mobility, fluctuation, pulsation, egg shell crackling, etc.
- Examine the teeth of lower jaw.
- Examine the cervical lymph nodes.
- Examine the temporomandibular joint for its movements.
- In case of discharging sinus of chin near midline, examine lower teeth for any sepsis (median mental sinus).
- In case of thickened, tender mandible with overlying sinus discharging dead bone pieces, it is due to chronic osteomyelitis.
- In case of painful swelling of the mandible following trauma examine for fracture.

Look for:

- ☐ Blood stained saliva.
- ☐ Difficulty in articulating words
- ☐ Laceration of the gums.
- ☐ Loss of continuity of lower border of mandible.
- ☐ Palpable crepitus.

26

Chapter

Imaging Techniques for Head and Neck Lesions

Sanjay Marwah

CONVENTIONAL RADIOGRAPHY

X-rays were discovered more than a century ago by Roentgen and are now used in all forms of conventional radiography as well as CT scan. Different types of tissues produce different degrees of X-ray attenuation depending on their density (Box 26.1).

Box 26.1: Tissue appearance on X-rays

Tissue	Appearance
Air (in lungs)	Black (Transparent)
Calcified tissue (bones)	White (Opaque)
Soft tissue (muscles)	Gray (intermediate transparent)
Fat	Dark gray (relatively more transparent)

Patient Positioning

Most X-rays are taken using standardized projections. Conventionally, these are described with respect to the direction of X-ray beam.

- Frontal views are taken with the patient's sagittal axis in line with the X-ray beam.
 - Lateral views are taken parallel to coronal axis.
 - A posteroanterior (PA) view is taken with tube behind the patient and the cassette (detector) placed touching the anterior surface of part to be X-rayed.
 - In an anteroposterior (AP) view, these positions are reversed. Due to direction of X-ray beam, anterior structures appear relatively larger on an AP film than on a PA film. Similarly posterior structures appear larger on a PA film.
 - Other projections are described with respect to the side closest to the cassette, e.g. in left lateral view, X-ray beam passes from right to left.
- X-rays of skull are done in AP and lateral views and are mostly indicated following trauma to detect fractures (see Fig. 17.2B) or to detect bone destruction (Figs 26.1A to C and 26.2).
 - Face and jaws present unusual problems in radiographic examination that are overcome with special projections (Also see Chapter 21: Fractures, Maxillofacial Fractures):
 - Posteroanterior view of mandible in open mouth position is done for body of mandible, ramus and neck of condyle.
 - Posteroanterior view of maxilla in Water's position is done for sinuses and zygomatico-maxillary complex.
 - True lateral view of skull for nasal bones.
 - Submentovertex projection for base of skull.
 - 'Jug handle' view of skull for zygomatic arches.
 - Radiograph for temporomandibular joint is done in closed mouth and open mouth position.
 - Intraoral projections include periapical films, occlusal films and bite wing films.
 - In orthopantomography, the position of object is fixed whereas the X-ray tube as well as film moves in a semicircular fashion. It covers a relatively large area of the jaws. The mandible is seen from condyle to condyle whereas maxillary region superiorly extends to orbital region. The only disadvantage is magnification and geometric distortion shown in this film.

ULTRASONOGRAPHY

Principle

Ultrasound is the name of high frequency sound waves above the limit of human audibility (> 20 kHz).



Fig. 26.1A: Clinical photograph showing chronic osteomyelitis vault



Fig. 26.1B: X-ray skull AP view showing bone destruction—osteomyelitis



Fig. 26.1C: X-ray skull lateral view showing bone destruction—osteomyelitis



Fig. 26.2: X-ray skull showing flea bitten areas—multiple myeloma

The ultrasound waves are generated by a piezo-electric transducer (probe) that is capable of changing electrical signals into mechanical (ultrasound) waves. These waves are transmitted in beams and are used to scan the body tissues. Different tissues alter the waves in different ways, some reflect while others scatter them (Box 26.2).

Thereafter the reflected waves return back to the transducer as echoes and are converted to electrical signals. These signals are reconstructed as a two-dimensional map of all the tissues that is displayed on

Box 26.2: Reflection of ultrasound waves by various tissues

- **Fluid** (urine, ascites): All the waves are allowed to pass without reflection.
- **Bone, Air** (lungs): All the waves are reflected back and not allowed to pass.
- **Soft tissues** (muscles, fat): Waves are partly reflected back and partly allowed to pass.

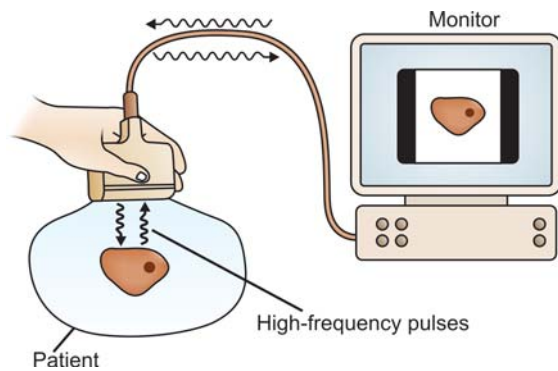


Fig. 26.3: Functioning of ultrasound

a video monitor (Fig. 26.3). Based on this display, various modes of ultrasound are:

1. **A-mode:** The echoes are shown as peaks.
2. **B-mode:** The image shows all the tissues traversed by the ultrasound scan.
3. **Real time:** The multiple B-mode images are watched in rapid sequence. The images change with each movement of the transducer or if any part of the body is moving (e.g. pulsating artery). It is possible to “freeze” any displayed image so that it can be studied carefully and can be measured.
4. **M-mode:** It is another way of displaying motions in form of a wavy line. It is most commonly used for cardiac ultrasound.

Doppler Ultrasound

It is used to detect and measure the rate of movement of any fluid such as blood. It is based on the principle of “Doppler effect”.

When ultrasound waves are transmitted towards a stationary reflector, the reflected waves (echoes) remain of same frequency as those of originally transmitted. However, if the reflector is moving towards the transducer (probe), the reflected frequency will be higher than the transmitted frequency. Conversely, if the reflector is moving away from the transducer, the reflected frequency will be lower than the transmitted frequency (Fig. 26.4). This phenomenon is called as “Doppler effect” and the difference between the frequencies is called “Doppler shift”.

In a *Color Doppler*, the direction of flowing blood is distinguished by different colors.

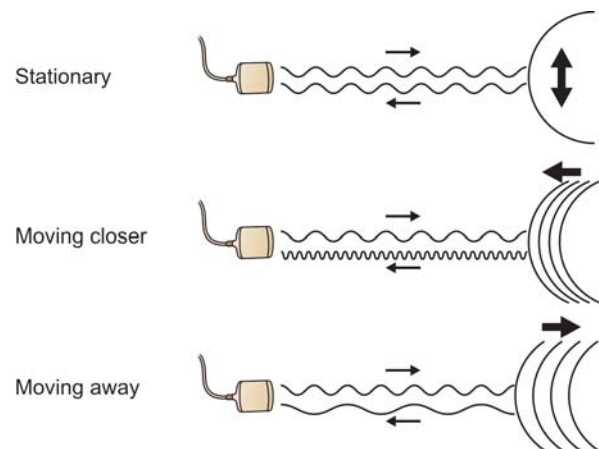


Fig. 26.4: Doppler effect

In a *Duplex Doppler system*, a blood vessel is located by B-mode ultrasound imaging and then blood flow is measured by Doppler ultrasound.

Terminologies

- **Acoustic beam:** The beam of ultrasound wave produced by the transducer (probe).
- **Anechoic (anechogenic):** Without echoes, e.g. normal urine and bile.
- **Hyperechoic:** It describes tissues that create brighter echoes than adjacent tissues, e.g. bone.
- **Hypoechoic:** It describes tissues that create dimmer echoes than adjacent tissues, e.g. lymph nodes, some tumors.
- **Internal echoes:** Ultrasound reflections from tissues of different density within an organ, e.g. gallstones within a gallbladder.
- **Acoustic window:** A tissue that offers little obstruction to the ultrasound waves and therefore used as a route to obtain images of deeper structures, e.g. bladder full of urine allows access to the pelvic organs.

Advantages of Ultrasound

- Outdoor procedure
- Noninvasive
- Painless
- Flexible
- Comparatively inexpensive
- Easily repeatable

- Does not depend on organ function
- No additional contrast is required.
- It requires no special patient preparation.

Drawback of Ultrasound

Since bones and air completely reflect the ultrasound waves, so deeper structures cannot be evaluated. Hence, it is not useful for detecting pathologies of head and chest (lungs contain air) due to lack of “acoustic window”.

ULTRASOUND FOR HEAD AND NECK LESIONS

The use of ultrasound evaluation is limited to following problems in the head and neck:

1. **Vascular abnormalities:** Ultrasound is an excellent screening test for evaluation of stenosis of large vessels in the neck particularly carotid arteries (e.g. due to atheromatous plaque). Color Doppler is particularly useful in such cases. Ultrasound is also very useful in determining whether a pulsatile neck mass originates from a vessel (e.g. carotid artery) or whether it is merely adjacent to and displacing nearby vessels.
2. **Eye lesions:** Ultrasound examination is valuable for evaluating the eye for the mass lesions of the globe itself. However, it is less effective for evaluating retro-ocular disease.
3. **Thyroid mass:** Ultrasound is most useful in differentiating solid from a cystic mass. The thyroid mass can be local or diffuse, single or multiple.

I. Focal masses

- a. **Solid mass:** 70% of focal lesions are solid thyroid nodules and these are mostly benign adenomas. Ultrasound can pick up a solid mass as hypo- or hyperechoic lesion but it is not possible to differentiate between benign adenoma and malignant tumor.
- b. **Cystic mass:** True cysts are rare and characteristically they are seen as circumscribed, echo free lesions.
- c. **Abscess/hemorrhage:** They appear as ill-defined cystic lesion with internal echoes.
- d. **Calcification:** It is commonly seen in adenoma but may occur in papillary carcinoma thyroid. It is seen as hyperechoic areas with distal acoustic shadowing.

II. Diffuse masses

- a. **Homogeneous enlargement:** It may be due to puberty goiter, endemic goiter, hyperthyroidism or acute thyroiditis. Enlargement is usually seen as a solid mass that is ultrasonically homogeneous.
 - b. **Heterogeneous enlargement:** It is commonly seen in multinodular goiter. Ultrasound shows heterogeneous enlargement of thyroid with multiple nodules, some of which may show cystic degeneration.
4. **Other neck masses:** Ultrasound can evaluate various masses in the neck and can demonstrate their relationship with the thyroid and major vessels. However, etiology of such masses is not always recognizable.
 - a. **Abscess:** The size and shape of the abscesses in the neck is variable. Their outline is irregular and internal echoes are seen.
 - b. **Lymphadenopathy:** The diagnosis of lymph node enlargement in neck is largely clinical. On ultrasound, lymph nodes appear as hypoechoic masses with regular outline, single or multiple, variable size, oval or round is shape. The cause of lymph node enlargement cannot be determined on ultrasound.
 - c. **Cystic hygroma:** On ultrasound, it is seen as fluid filled (anechoic) and septate mass. It is variable in size and may extend in thorax or axilla and the extension can be picked up on ultrasound examination.
 - d. **Cysticercosis:** It is seen as a circumscribed cystic mass in the neck muscles with echogenic nidus in the cyst.
 - e. Other masses in the neck like lipoma, dermoid, thyroglossal cyst, branchial cyst, hematoma can be seen on ultrasound.
 5. **USG guided FNAC:** Ultrasound is very useful tool in guiding the placement of needle for FNAC that helps in determining the pathological diagnosis of neck masses.

CT SCAN

Computed tomography (CT) imaging has made very important medical breakthrough and its inventor, Godfrey Hounsfield received a Nobel Prize in 1979. CT became possible because of developments in computer technology.

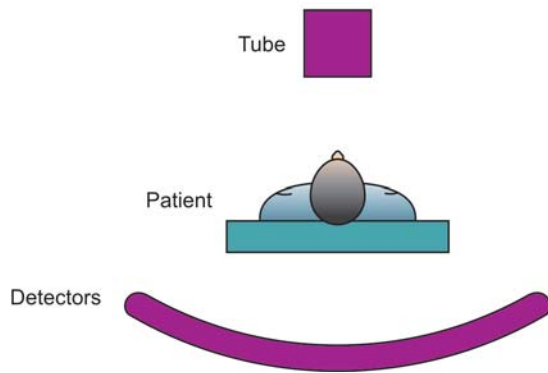


Fig. 26.5: Typical CT configuration

Principle

An X-ray tube rotates around the patient who is placed in the center of the scanner (Fig. 26.5). A series of thin X-ray beams are passed through a chosen transverse plane of the body and a diametrically opposed detector measures the extent of its absorption. The measurements are fed into a computer that generates an X-ray picture of the transverse section of the body displayed on television monitor.

Early CT scanners took several minutes to acquire a single slice. Modern scanner can acquire a single slice in a second or less by use of sophisticated arrangements of detectors and tube systems and more powerful computers. Slices of 1-2 mm width provide very good detail of the tissues.

Spiral (Helical) CT

It uses a 'slip ring' technique whereby the X-ray tube can rotate continuously around a fixed ring of detectors. The patient is moved in and out of the gantry rapidly while scanning continuously (Fig. 26.6). Thus, entire

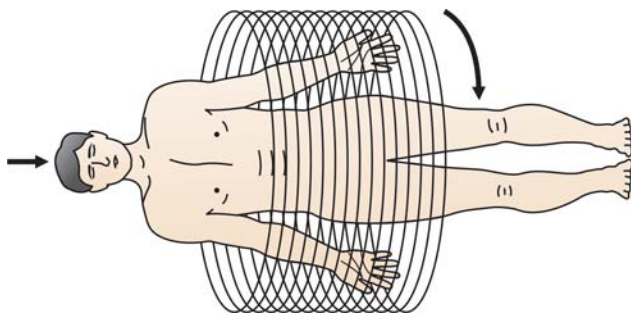


Fig. 26.6: Spiral (Helical) CT

study can be performed during a single breath hold. Thus, instead of reconstructing serial axial sections (as in a conventional scan), spiral CT produces a three dimensional picture. Spiral CT has many advantages over conventional or axial CT, including:

- It minimizes motion artifacts.
- It reduces patient dose.
- It improves spatial resolution by giving three-dimensional picture.

Multislice or Multidetector Spiral CT

When spiral CT has added dimension of multiple arcs of detectors, it is known as multidetector spiral CT. Its advantage is that a volume of contiguous slices as thin as 0.625 mm can be obtained within seconds, decreasing motion artifacts and the need for patient sedation and improving 3D representation.

Reading a CT Film

Unlike conventional radiography, CT is a digital modality. A typical CT image can be considered as matrix of elements (pixels). Each pixel has a gray scale intensity volume that represents X-ray attenuation (tissue density). X-ray attenuation values are scored from -1000 to + 1000 Hounsfield Units (HU). By convention, air is - 1000 HU and water is 0 HU. Attenuation values can be used to differentiate tissues and to analyze some types of pathologies.

- Fat and gas have negative attenuation values.
- Cysts and necrotic areas have values close to that of water (0 HU).
- Bone has high attenuation value (300-1000 HU).

Thus, fat represents an excellent contrast medium and the best scans are obtained on patients with an adequate amount of fat. Conversely, it is difficult to obtain good scans on emaciated patients with less fat.

Use of contrast agents can artificially increase the local attenuation and help in increasing the resolution of CT scan. Thus, intravenous contrast (iodine) will enhance blood vessels and oral contrast (barium) will delineate bowel.

Advantages of CT Scan

- It gives three-dimensional image of the body.
- Contrast resolution of CT is much better than conventional radiographs.

- CT delineates various body structures from each other and demonstrates their relationship.

Drawbacks of CT Scan

- Expensive investigation.
- Side effects of ionizing radiations, since CT depends on the use of X-rays.

CT SCAN FOR HEAD AND NECK LESIONS

CT scan has revolutionized the investigative approach to intracranial pathology. Various lesions that can be picked up on **CT head** are:

a. Vault and Skull Base Lesions

- Depressed fractures
- Osteolytic lesions
- Hyperostosis

b. Intracranial Lesions

- Tumors (primary, metastatic)
- Trauma (contusion, hematoma)(see Figs 17.7 to 17.9)
- Abscess
- Granuloma
- Infarction

On CT film, look for the “site” of the lesion and whether the lesion is within or outside the brain substance.

Look for the “mass effect” produced by the lesion, viz.:

- Ventricular compression
- Midline shift
- Obliteration of basal cisterns, sulci.

Look for the attenuation value (density) of the lesion, viz.:

i. High density lesions

- Blood
- Calcification (tumor, AV malformation, aneurysm, hamartoma)

ii. Low density lesions

- Tumor
- Abscess
- Edema
- Infarction
- Cyst
- Resolving hematoma

iii. Mixed density lesions

- Tumor
- Abscess
- Contusion
- Hemorrhagic infarct
- AV malformation

Some of the lesions appear only after contrast enhancement, e.g. vascular lesions.

Head and Neck Tumors

- CT scan is highly sensitive in detecting cortical bone destruction by tumor.
- In defining exact extent of intraoral and paranasal sinus tumors in difficult to examine areas such as parapharyngeal space, larynx and nasopharynx.
- It gives three-dimensional picture of the tumor and demonstrates tumor relationship with the adjoining normal structures.
- In parotid gland, CT is helpful in determining extension of tumor into the deep lobe.
- CT is an effective means of guiding the placement of a needle for biopsy or for percutaneous treatment of a lesion in neck or base of skull.

Traumatic Injuries

Fractures of head and facial skeleton occurring after trauma are best detected with CT scan (see Fig. 17.1). Before CT evaluation of facial fractures, cervical spine injury should be ruled out both by clinical and imaging methods.

Evaluation of foreign bodies is done with CT scan. However, it needs thin sections (1.5 mm).

Inflammatory Lesions

CT scan is usually performed for suspected inflammatory lesion when it does not respond to conservative therapy and surgical intervention is planned. CT scan evaluates the extent of paranasal sinus inflammatory disease and also defines the extent of osteomyelitis.

Cervical Adenopathy and Neck Masses

CT scan provides accurate anatomical location of the neck mass and its relation to adjacent vascular, muscular and neural structures. Although exact tissue diagnosis is not always possible, but careful analysis of imaging features of neck mass combined with clinical history and

physical examination gives a reasonable diagnosis in most cases.

a. *Nodal Neck Masses*

- Normal lymph nodes are often invisible on CT scan and they typically measure less than 1 cm.
- Any node measuring more than 1.5 cm in diameter is abnormal.
- Any node with central lucency, regardless of size is abnormal.
- Obliteration of fascial planes around a node is abnormal.
- CT scan is very useful in evaluation of metastatic neck nodes and helps in accurate staging of the malignancy. It is particularly useful when examination of neck is difficult because of obesity, previous surgery or radiotherapy.
- Presence of multiple nodes with a variable CT appearance (homogeneous, necrotic, enhancing, calcified) is most compatible with granulomatous disease (e.g. Tuberculosis).

b. *Non-nodal Neck Masses*

I. *Developmental masses*

- *Branchial cleft cyst*: Seen as well-circumscribed, unilocular, low density mass adjacent to sternomastoid muscle.
- *Cystic hygroma*: Poorly circumscribed, multilocular, low density lesion without peripheral rim enhancement seen in posterior triangle of neck.
- *Thyroglossal cyst*: Well-circumscribed, low density mass in midline of neck anteriorly.
- *Dermoid cyst*: Mass with peripheral rim enhancement, containing fat and fluid components seen in midline.

II. *Inflammatory masses*: Abscesses appear as single or multilocular low density masses that conform to fascial spaces. In contrast films, there is peripheral rim enhancement.

III. *Vascular masses*

- CT scan differentiates between pulsatile masses and aneurysms of cervical arterial system.
- Internal jugular vein thrombosis is seen as less dense area on contrast enhancement than blood.

- Paraganglioma (carotid body tumor) on CT scan shows intense enhancement after intravenous contrast injection.

IV. *Neurogenic masses*: Schwannomas and neurofibromas appear as hypodense or isodense to skeletal muscle on non-contrast CT. On giving contrast, enhancement pattern of neural tumors is highly variable (intense enhancement to lack of enhancement).

V. *Mesenchymal masses*:

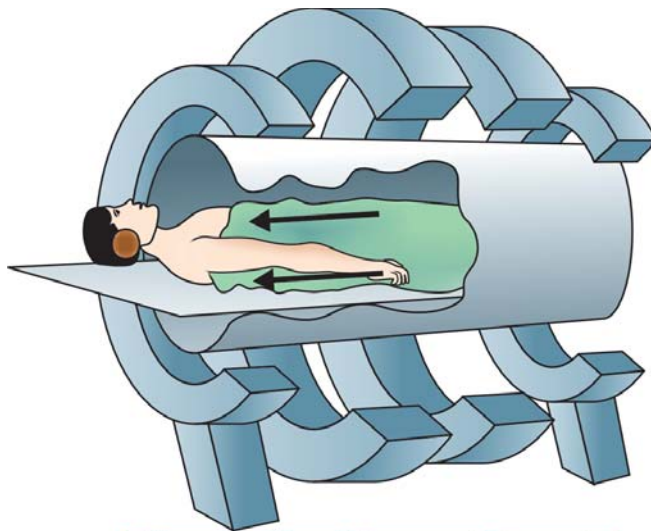
- Lipoma is seen as homogeneous non-enhancing mass isodense with subcutaneous fat.
- Malignant mesenchymal tumors (rhabdomyosarcoma) tend to destroy bone and distort soft tissue planes.

Masses Arising from Aerodigestive Tract

- These masses are cervical extension of diseases arising from oral cavity, larynx or hypopharynx.
- Ranula is seen as thin walled, unilocular, homogeneous cystic lesion in the floor of mouth.
- Laryngocele is seen as thin rimmed, fluid filled or air filled mass lateral to thyrohyoid membrane continuing with larynx.
- Pharyngocele also herniates through thyrohyoid membrane but is continuous with pyriform sinus rather than laryngeal ventricle.

Thyroid and Parathyroid Glands

- Ultrasound is often the first modality used to image thyroid gland because it detects more thyroid masses than CT or MRI.
- Main role of CT scan is in evaluation of thyroid malignancies. Its advantages are:
 - Evaluation of thyroid capsule transgression.
 - Detection of neoplastic infiltration into adjacent structures including ribbon muscles, carotid sheath and aerodigestive tract.
 - Identification of malignant lymphadenopathy.
- In parathyroid glands, since location of inferior parathyroid glands varies, advantage of CT scan is that it evaluates entire neck and mediastinum from skull base to aortic arch. However, distinction between lymphadenopathy and parathyroid adenoma is difficult on CT scan. CT guided FNAC



The patient lies within a powerful magnet. The magnetic field is aligned with the patient

Fig. 26.7: Position of the patient during MRI

may be used to sample parathyroid gland enlargement.

MAGNETIC RESONANCE IMAGING (MRI)

Principle

It is based on the magnetic properties of hydrogen nucleus. In an MRI examination, the patient is placed in a powerful magnetic field with which the protons within the body become aligned (Fig. 26.7). Radiowaves in the form of a radiofrequency pulse transmitted into the patient cause the alignment of the protons to change (e.g. by 90°). When this radiofrequency pulse is turned off, the protons in the patient's body return to their neutral position, emitting their own weak radiosignals which are detected by receiver coils and used to produce an image using powerful computers (Fig. 26.8).

T_1 and T_2 Weighted Images

Two independent relaxation rates are usually described with respect to the direction of main magnetic field:

- Longitudinal relaxation or T_1 relaxation time. Long T_1 times reflect slower relaxation parallel to the main magnetic field. In T_1 weighted images, areas with long T_1 time give a low signal, i.e. more black.
- Transverse relaxation or T_2 relaxation time. Long T_2 times reflect slower relaxation in the transverse

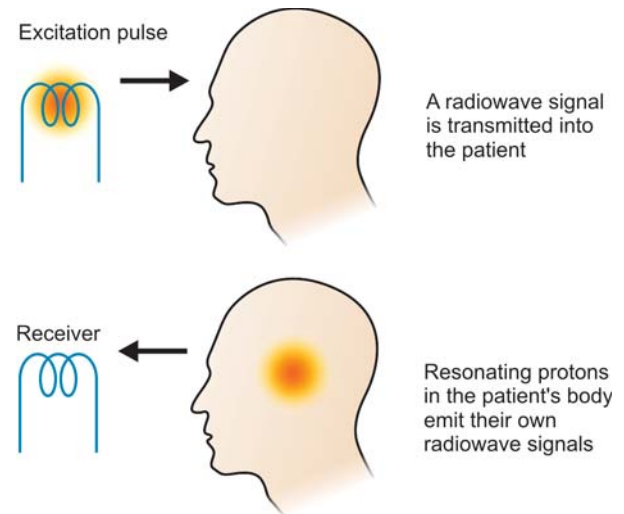


Fig. 26.8: Basic principles of MRI

plane. In T_2 weighted images, areas with long T_2 time give a high signal, i.e. more white.

Tissues with high water content have particularly long T_1 and T_2 times and therefore appear dark on T_1 weighted images and bright on T_2 weighted images. Similarly, there are large natural differences between different soft tissues and these differences are exploited in outlining these tissues (Box 26.3).

Box 26.3: T_1 and T_2 relaxation times

Tissue	T_1 (milliseconds)	T_2 (milliseconds)
Gray matter	520	95
White matter	380	85
CSF	1500	1000
Skeletal muscles	250	50

Radiofrequency Sequences

An MRI sequence consists of a series of excitatory radiofrequency pulses with a particular orientation to main magnetic field. Important sequences include:

- Spin echo sequence
- Gradient echo sequence
- Echoplanar imaging
- Inversion recovery sequence

Contrast Agents

Intravenous chelated **gadolinium** reduces T_1 relaxation time and thus areas of enhancement appear bright on T_1 weighted images. It is helpful in vascular lesions and areas of increased permeability (e.g. tumor neovascularization). It also helps in differentiating tumor tissue from surrounding edema. It has an excellent safety profile.

MRI vs CT SCAN

- A conventional MRI scan comprises an array of small picture elements (pixels) as in CT but contrast resolution of MRI is at least 100 times better than CT.
- In CT, soft tissue characterization depends on only one variable—electron density.
- In MRI, soft tissue characterization depends on many variables—proton density, relaxation times of tissue protons (T_1 and T_2 relaxation times), water content, protein content, composition of various tissues (fat, muscles, fibrous tissue, etc.), vascularity.
- Resolution of high density objects, e.g. cortical bone is better with CT than MRI, because there is essentially no MRI signal from dense cortical bone. So cortical bone destruction is better seen with CT than with MRI.
- MRI is excellent in detecting fat containing marrow within body structures. Hence, infiltrating disorders of the bony skull base can be evaluated with MRI on the basis of the infiltration of bone marrow rather than on bone destruction.
- The ability of MRI to differentiate a nerve from fat by use of fat suppression techniques and from CSF makes MRI the procedure of choice to visualize the anatomy of cranial nerves above and below the skull base and to define its involvement with a disease process.
- CT images are usually obtained in the axial plane while MRI can acquire images in three planes (coronal, sagittal, oblique).
- For contrast enhancement, CT uses iodinated contrast that can be problematic in patients allergic to the contrast agent. MRI is excellent replacement to CT in such patients since it is able to differentiate soft tissues without use of the contrast.
- Unlike CT scan, MRI does not use ionizing radiation that can be a problem for the lens of the eye.

Advantages of MRI

- No bone artifacts.
- No ionizing radiations.
- Better differentiation of fluid from soft tissues.
- Can select any plane for imaging.

Drawbacks of MRI

- Limited slice thickness—3 mm (1 mm in CT).
- High cost.
- Powerful magnetic fields can dislodge or interfere with some medical devices (e.g. pacemakers, ferromagnetic aneurysm clips). These are absolute contraindications.
- Claustrophobia.
- Bone imaging limited to display of marrow.
- Tissues containing low number of suitable hydrogen nuclei (e.g. aerated lungs, dense bone) are poorly visualized. In such areas, CT is better investigation.

MRI for Head and Neck Lesions

It is indicated in following situations:

- To define the extent of soft tissue tumor and its relation with adjoining soft tissues or fluids.
- To detect perineural tumor spread and intracranial infiltration.
- To distinguish fluid from tumor in an obstructed paranasal sinus.
- To evaluate all cranial neuropathies.
- To detect meningeal disease of head and spine from inflammation or tumor.
- To evaluate possibility of recurrent tumor.
- To evaluate possibility of cartilage erosion by tumor.
- To evaluate larynx particularly due to ability to define submucosal tissue planes.
- To evaluate areas where artifacts may degrade the CT image, e.g. due to dental amalgam in mouth.
- To evaluate relationship of major blood vessels to a soft tissue tumor.
- To evaluate temporomandibular joint.
- *Magnetic resonance angiography*: Traditional angiography is invasive procedure and requires selective placement of catheter into the arteries of head and neck. However, in MRI, the movement of excited protons within the vessels appears as areas of high or low signals and can be exploited to produce noninvasive angiographic images.

- **Interventional procedures:** Although most image guided procedures of the skull base and neck are performed with CT, MRI guidance has advantage that biopsy needle can be placed into a definite lesion that cannot be easily detected even on enhanced CT. Such MRI-guided biopsy techniques require use of open MRI system and MRI compatible needles (non-ferromagnetic needles) that have been developed recently.

CT and MRI are complementary in following situations:-

- Diseases of lymph nodes.
- Congenital anomalies of head and neck.
- Areas having significant natural contrast because of fat, muscle, bone and air, e.g. infrahyoid neck, orbit.
- In evaluation of suprahyoid neck, oropharynx and nasopharynx.

INTERVENTIONAL RADIOLOGY

Under radiological guidance (image intensifier, USG, CT guided) invasive procedures are performed for diagnostic as well as therapeutic purpose.

Its types are: Vascular and non-vascular.

Vascular

- **Angiography:** The arterial system is outlined by injecting contrast medium into the vessel lumen, so as to define various lesions, e.g.
 - Aneurysm is seen as focal dilatation.
 - Vascular occlusion is seen as blockade in the vessel lumen with or without collaterals.
 - Tumor vessels are seen as neovascularization (tumor blush).
 - Arterio-venous malformation is seen as dilated arteries with rapid drainage into veins.
- Technique:** See Chapter 18: Gangrene and Diseases of Arterial System.
- **Angioplasty:** Under image intensifier (X-ray screening device), blocked arteries are dilated using balloon catheters, e.g. carotid, coronary, aorta, renal arteries, etc.
- **Stenting:** After dilatation of the artery, a tubular stent can be placed at the site of blockage so that arterial lumen remains patent.
- **Catheter insertion:** Under guidance, catheter is inserted into a vessel for following purposes:

- **Therapeutic embolization:** In a deep seated bleeding vessel, it helps in controlling the bleed and avoids open surgery, e.g. massive hematuria due to vascular erosion by renal cell carcinoma, GI hemorrhage due to mesenteric vascular bleed. The site of bleeding is identified by angiography and then a foreign substance (spring coil, gel foam, etc.) is injected into bleeding vessel so as to block it.
- Injection of a clot lysing agent to dissolve a blood clot blocking a vessel so that blood flow is restored, e.g. coronaries, cerebral vessels.
- To deliver chemotherapy drugs locally at the site of tumor. It helps in increasing efficacy and decreases toxicity of the drug.

Non-vascular

- USG guided biopsy from lesions in deep seated organs, e.g. liver, prostate.
- Percutaneous drainage of obstructed organs, e.g.
 - Percutaneous nephrostomy in blocked kidneys (hydronephrosis).
 - Percutaneous biliary drainage in malignant obstructive jaundice blocking biliary system.
 - Percutaneous drainage of deep seated abscess (liver abscess).
 - Percutaneous feeding gastrostomy in comatose patient.

The main advantage of interventional procedures is that it gives good palliation with minimum increase in morbidity and mortality. It also avoids open surgery in unfit patients.

RADIONUCLIDE STUDIES

PET Scan

Positron emission tomography (PET) is a technique that can detect a number of positron emitting radionuclides and therefore can be used to study a variety of metabolic processes in an organ or lesion.

F-18 fluoro-2-deoxy-D-glucose (**F-18 FDG**) is the most commonly used positron emitting radiopharmaceutical used for PET imaging.

Conventional imaging relies on morphological changes; whereas, PET being a functional imaging modality detects the disease process in its early phase

as metabolic abnormalities that generally precede anatomical changes.

However, apart from being expensive technology, PET scan is limited in its ability to provide information on the exact localization of lesions because of the absence of precise anatomic landmarks.

PET used in conjunction of CT scan (**PET-CT**) is more useful as it provides anatomical details with CT scan overlapping with the abnormal uptake with PET scan. It is a single-gantry hybrid system with the patient passing directly from a PET scanner to a CT scanner without moving from the table.

After completion, matching pairs of PET and CT images are fused and are seen in axial, coronal and sagittal planes.

Role of PET-CT is in head and neck oncology for detecting and grading tumors, monitoring response to therapy, distinguishing between residual tumors and post-treatment scarring and recurrent tumors.

SPECT

Single-photon emission computed tomography technique uses compounds labeled with gamma emitting tracers but unlike conventional scanning, acquires data from multiple sites. The price of SPECT study is less than PET study and is competitive with CT and MRI. Unfortunately, SPECT study suffers from relatively low spatial resolution so that they are not effective in detecting subtle abnormalities like small tumor recurrences. The SPECT image is examined in conjunction with CT or MRI image (structural image) to aid interpretation.

27

Chapter

Burns and Skin Grafting

Sanjay Marwah

BURNS

Definition

It is an injury or damage caused by heat or sources producing heat leading to coagulation necrosis.

- Damage rarely occurs when temperature is below 45°C.
- At temperature more than 50°C, protein denaturation occurs in cells.

Etiology

Various causes of burns are:

1. **Dry heat:** It is caused by fire from coal, cooking gas, kerosene, petrol, etc.
2. **Wet heat:** It is caused by hot liquids, e.g. boiling water, tea, coffee, etc. and the injury is known as *scald*. These are usually minor burns.
3. **Electric burns:** Electric current causes burns as well as systemic complications (Box 27.1).
 - There is a point of entry where current touches the body.
 - The current passes through tissues causing tissue damage.
 - The current leaves the body at grounded area.
4. **Chemical burns:** It is due to acids or alkali. It causes progressive damage because the agent remains in contact with the skin and chemical injury continues.

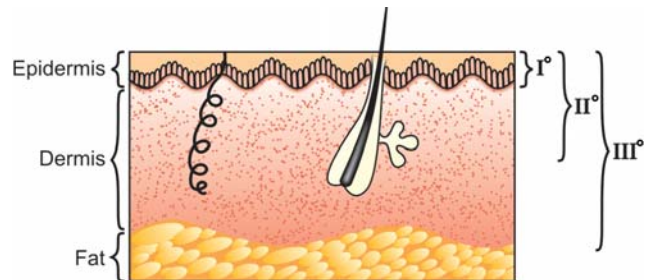


Fig. 27.1: Section of skin showing various degrees of burns

5. **Irradiation:** It is due to radiotherapy given for cancer treatment. It causes radiodermatitis.

Degrees of Burns

The skin consists of superficial layer (epidermis) and deep layer (dermis). There are appendages in skin known as *pilosebaceous elements*. These lie in the dermis but project in epidermis as well. These are hair follicles, sweat and sebaceous glands.

Burns are divided into three degrees based on thickness of involved skin (Fig. 27.1):

I° burn: It is a burn of epidermis only, e.g. sunburn.

II° burn: It is a burn involving epidermis and part of dermis, e.g. flashburns, scalds.

III° burn: It is a burn involving epidermis and full thickness of dermis, e.g. flame, chemical and electric burns.

Box 27.1: Systemic complications of electric burns

Heart	- Cardiac arrhythmias	
Blood vessels	- Thrombosis	→ Gangrene
Muscles	- Myoglobinuria (port-wine urine)	→ Renal failure



Fig. 27.2: Second degree burns

Clinically

Various degrees of burns present as follows:

I° burn: There is painful erythema of skin. It is tender to touch and blanches on pressure. It is of little clinical significance because water barrier of skin is not disturbed. Hence, it is not considered while estimating magnitude of burn injury and planning fluid replacement.

II° burn: It usually presents with painful blisters. When blisters rupture, the surface of burn appears red, shiny and wet (angry looking) (Fig. 27.2). It is due to cutaneous edema.

III° burn: It is painless because all cutaneous nerves are burnt. The burn surface appears dry, charred, grayish black in color. There is no cutaneous edema (Fig. 27.3).

II° burn may readily be converted to III° burn because of infection leading to destruction of residual pilosebaceous elements.

Healing

Healing in various degrees of burns takes place as follows:

I° burn: Healing occurs by regeneration of remaining pilosebaceous elements that form epidermis. Healing takes place in 3-5 days and there is no scarring.

II° burn: Epidermis grows gradually from the ends of remaining pilosebaceous elements on surface. Healing takes place in 7-14 days with minimal scarring.



Fig. 27.3: Full thickness burns

III° burn: Since no pilosebaceous element is left so burnt area can not heal by itself. Small burn area can heal by scarring in 3-5 weeks while a larger area requires skin grafting.

Management of Burns

First Aid Measures

These are the measures required at the site of accident (Box 27.2).

Box 27.2: First aid measures for burns

- Remove patient from burning agent.
- Pour water on body to extinguish fire.
- Put off electricity if electric current is involved.
- In chemical burns, wash the skin surface.
- In case of indoor fire, remove patient from smoke filled room.
- Apply cold or lukewarm water on burnt area.
- Advantages of pouring water are:
 1. Extinguishes fire
 2. Easily available
 3. Less fluid evaporation from surface
 4. Reduces pain.
- Wrap the patient in clean dry sheet and shift to hospital.

Emergency Management

Initial assessment and management of burn patient is as per ATLS guidelines (See Chapter 10: Care of the Acutely Injured).

a. Airway

- History of flame burns suffered in closed space can cause smoke inhalation due to respiratory burns. Therefore signs of airway obstruction must be looked for.
- Burns of mouth, lip and neck result in soft tissue swelling occurring within hours of injury that causes sudden airway obstruction.
- In case of airway obstruction, endotracheal intubation or tracheostomy may be required.

b. Breathing

- Smoke inhalation is a major cause of mortality in burns.
- Heat causes damage to upper airway (oral cavity, nasopharynx, larynx).
- Toxic chemicals present in smoke like carbon monoxide cause damage to lower bronchial tree and lung parenchyma.
- Patient presents with hoarseness, stridor, wheezing and production of large amount of carbonaceous sputum.
- There is tachycardia, cyanosis and bronchospasm.
- Patient requires humidified oxygen, bronchodilators, steroids and artificial ventilation.

c. Circulation

- The patient needs rapid intravenous fluid replacement to compensate for the fluid lost from burn surface area.
- Since peripheral veins are usually not visible due to limb burns, so venesection (cut down) is done in the arm or leg to start intravenous fluids.
- Requirement of intravenous fluid replacement is calculated from percentage area of burns.
- If burn area is $> 15\%$ in an adult and $> 10\%$ in a child, it requires intravenous fluid replacement.
- Percentage area of burns** is calculated by following formulae:
 - Wallace rule of nine:** The body is divided into eleven parts and each part covers 9%, making it $11 \times 9 = 99\%$. The remaining 1% is the perineum (Fig. 27.4).
 - Hand method:** Size of patient's hand is taken as 1%. It is useful in calculating patches of burns.
 - Lund and Browder Chart:** In this chart, each part of body is given different percentage. Also at different age, different percentages are given.

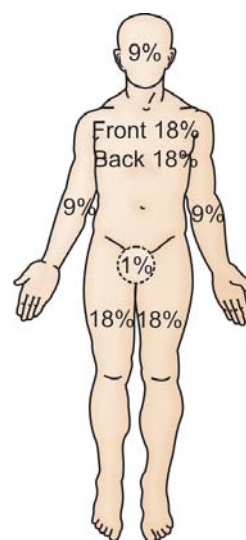


Fig. 27.4: Wallace rule of nine for calculating percentage area of skin burns

It makes more accurate assessment of burn area. Copies of such chart should be available in accident and emergency department for rapid calculation of burnt area.

- Fluid calculation** is done using one of the following formulae:
 - Parkland formula:** 4 cc/% burn/kg body weight of Ringer lactate is given in first 24 hrs. Half of the calculated fluid is given in first 8 hrs. 1/4th fluid is given in second 8 hrs. 1/4th fluid is given in third 8 hrs. Time for giving I/V fluids commences at the time of injury and not at admission to hospital.
 - Muir and Barclay formula** (Box 27.3):

Box 27.3: Muir and Barclay formula

$\frac{\text{Total \% of burn area} \times \text{body weight (kg)}}{2} = \text{Volume of fluid (ml) to be given in each time period}$
Time periods: $\underbrace{0-4, 4-8, 8-12}_{4 \text{ hourly}}, \underbrace{12-18, 18-24}_{6 \text{ hourly}}, \underbrace{24-36}_{12 \text{ hourly}}$ hours

- The formulae are only a guide and patient may require more fluid based on clinical condition.
- Fluid requirement is best guided by *urine output* that should be 30-50 ml/hr in an adult. If urine output decreases, increase the fluid supplement.

- d. *Other injuries* may be life-threatening (e.g. thoracic, abdominal, spinal) and should be dealt with appropriately.

Early Treatment

Oxygen therapy: Give 100% humidified oxygen via a face mask if inhalation injury is suspected.

Antibiotics: Start with basic antibiotics, e.g. penicillin to take care of gram-positive organisms. C-penicillin 10 lakh units is given 6 hrly in adults. Later on if complications occur, give broad spectrum antibiotics.

Tetanus prophylaxis: Check the patient's immune status and if in doubt give 0.5 ml tetanus toxoid I/M injection.

Analgesia: Partial thickness burns are extremely painful and require analgesia and sedation. Intravenous morphine (10 mg) or pethidine (50 mg) is given slowly and small increments are made till pain is relieved. It can cause nausea, vomiting and respiratory depression. Intramuscular injections are not effective because drug is not absorbed due to peripheral vasoconstriction caused by hypovolemic shock.

Catheterization: Aseptic urethral catheterization is done with Foley's catheter to maintain record of hourly urine output.

Antiulcer drugs: There is a risk of formation of gastric ulcers (Curling's ulcers) in burns due to stress causing hyperacidity. Hence, prophylactically, H_2 blockers (ranitidine-150 mg BD) or proton pump inhibitors (omeprazol-20 mg BD) should be given.

Nasogastric intubation: In first 24 hrs, there is risk of vomiting and aspiration pneumonia. The causes of vomiting are:

- Severe pain
- Narcotic analgesics
- Reflex paralytic ileus
- Gastric ulcers (Curling's ulcers)

Hence, early placement of nasogastric tube decompresses the stomach. Also, gastric contents can be inspected for any ongoing hemorrhage due to gastric ulcers. Later enteral feeding is started in severely burnt patients. It protects mucosal integrity and reduces risk of bacterial translocation and sepsis.

Monitoring

- Accurate input-output chart is maintained.

- In initial stages, temperature, pulse and respiration are recorded hourly.
- Blood pressure recording is usually not possible due to limb burns.
- Hourly urine output monitoring.
- In extensive burns (>60%), invasive monitoring should be done viz. central venous pressure monitoring, arterial line for blood gas estimation and Swan-Ganz catheter to record pulmonary wedge pressure.

Local Treatment

- Dressing of burn wound is done once vital signs are settled and analgesic injections are given.
- Dressing is not to be done immediately at time of admission because burnt area is very painful and there can be neurogenic shock (patient is already in hypovolemic shock).
- Dressing can be done in two ways:

- i. **Closed method:** The burnt area is cleaned with sterile saline and a local chemotherapeutic agent is applied followed by a layer of non-sticking gauze (vaseline gauze). It is covered with thick padding followed by light bandage. The dressing is changed when it becomes soaked or when inspection of the burn wound is required.

Advantages:

1. Closed dressing relieves pain.
2. Thick padding absorbs edema fluid.

Disadvantages:

1. Thick padding rises body temperature and causes fever.
2. If padding gets soaked up to surface, bacteria will enter in by capillary action causing burn wound sepsis.

- ii. **Open method:** The burn wound is cleaned with saline and left open after application of a chemotherapeutic agent. Exposure to light prevents bacterial proliferation. The surface gets dried up producing a crust of dry plasma.

Advantages: It is useful in parts difficult to dress, e.g. face, neck, perineum, buttocks.

Disadvantages:

1. It is difficult to manage asepsis and requires careful monitoring.
2. There is enormous loss of fluids and electrolytes from the exposed surface.

So, best method of dressing is to combine two methods:

Closed method—applied all over the body.

Open method—on face and genitals.

- **Local chemotherapeutic agents:** Aim is not to sterilize the wound but to decrease bacterial population so as to prevent burn wound sepsis.

Various agents used are:

Soframycin

Neosporin

Povidone iodine

Silver sulphadiazine.

Most commonly used agent is *silver sulphadiazine* and it is the best agent (Box 27.4).

Box 27.4: Silver sulphadiazine as local chemotherapeutic agent

Advantages

- Effectively penetrates *eschar**
- Non-toxic
- Non-allergic
- Effective against most organisms
- Soothing

Disadvantages

- Costly drug
- Emergence of opportunistic infection

**Eschar*: Layer of dead tissue formed on burnt surface due to coagulation of cell proteins.

Surgical Treatment

Escharotomy: Circumferential third degree burns anywhere in the body can cause secondary damage by their constricting effect. On the chest and neck, there can be breathing difficulty and tracheal compression respectively. In the limb, there can be decreased circulation leading to ischemia and gangrene. So, constricting portion should be quickly incised along the affected limb until eschar splits open and tension is relieved. It is done without anesthesia because there is no pain in third degree burns.

Debridement and excision: In 2° burns, the blisters are punctured and nonviable skin removed. It allows application of the drug directly to the wound.

In 3° burns, eschar remains tightly adherent to underlying tissues and cannot be separated without severe pain and bleeding. So, only loose eschar is

removed initially. In about 3 weeks time, eschar separates because of bacterial proteases. At this stage, it should be promptly debrided to prevent systemic sepsis.

Early tangential excision: It is done within 48 hrs of deep burns. Thin layers are removed till viable tissue is reached that is judged by capillary bleeding. The resulting wound is closed primarily or covered with skin graft.

Advantages:

1. Decreased period of pain and hospital stay.
2. Improves functional outcome.
3. Reduced scarring.

Disadvantages: Major surgical procedure requiring general anesthesia in already critical patient.

Delayed skin grafting: Once area of full thickness burns is adequately prepared with dressings and debridement (in about 3 weeks time) skin grafting is done to cover the defect.

Nutrition

In burn patients, there is continuous catabolic state due to skin loss. So, all patients need nutritional support. In most patients supplements are given orally. In significant burns, continuous feeding is done through nasogastric tube. The recommended food items are:

For vegetarians: Milk, pulses, banana, groundnut.

For non-vegetarians: Eggs, meat, fish, chicken.

Sometimes parenteral nutrition is required in case of systemic sepsis.

Complications of burns are given in Box 27.5.

Box 27.5: Complications of burns

- Smoke inhalation syndrome
- Hypovolemic shock
- Septic shock
- Neurogenic shock
- Renal failure
- Electrolyte imbalance
- Curling's ulcers—hematemesis
- Gastric ulcer—perforation peritonitis
- Suppurative thrombophlebitis (at site of I/V lines)
- Malnutrition (protein loss)
- Hypertrophic scars/ contractures
- Marjolin's ulcer
- Suppurative chondritis (thermal injury to ear cartilage)

SKIN GRAFTING

It is a technique in which skin is transferred from one site to another site.

Donor Site

It is the area from where skin is taken.

Recipient Site

It is the area on which skin is transferred. It should be clean, free from infection and with healthy granulation tissue at the time of grafting so as to prevent graft rejection (Fig. 27.5).



Fig. 27.5: Recipient area with healthy granulation tissue

Methods of Grafting

Skin can be transferred in two ways:

a. Grafting

Independent transfer of skin from donor area to recipient area without maintaining continuity so that all vessels nourishing the graft are cut.

b. Flap

The blood supply of skin is maintained by a pedicle during transfer.

Depending upon the thickness, skin grafts are of two types (Box 27.6).

i. Split Thickness Graft (STG)

Split thickness graft (STG) consists of epidermis and variable part of dermis (Fig. 27.6). Its types are:

Box 27.6: Types of skin graft

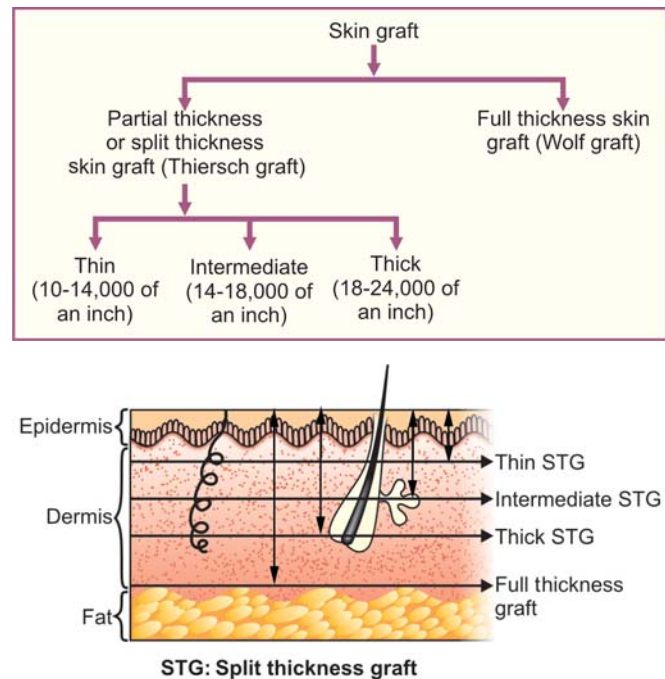


Fig. 27.6: Diagrammatic representation of graft thickness

Thin STG: It has epidermis and very thin layer of dermis. It is used for resurfacing large wounds, e.g. post-burn wound.

Its *advantages* are:

- ☐ Survival of the graft is very good.
- ☐ Large quantity can be taken from the body.

Its *disadvantages* are:

- ☐ The graft contracts after application leading to post-operative deformity.
- ☐ Cosmetic result is poor.

Intermediate STG: It has epidermis and half thickness of dermis.

Its *advantages* are:

- ☐ Large quantity can be taken from the body.
- ☐ Cosmetic result is better than thin STG.

Its *disadvantages* are:

- ☐ Survival of graft is poorer than thin STG.
- ☐ Cosmetic result is poorer than full thickness graft.

Its *indications* are: Large raw areas with clean base, e.g. after tumor excision, after release of contractures.

Thick STG: It has epidermis and major part of dermis. It is rarely used. It has better cosmetic results. So, it can be used on exposed body parts with large raw areas.

ii. Full Thickness Graft

It consists of epidermis and full thickness of dermis without any fat (Fig. 27.6).

Its *advantages* are:

- Very good cosmetic results.
- Postoperative deformity is less.

Its *disadvantages* are:

- It does not survive well.
- Large quantity cannot be taken.
- Donor area needs wound closure or cover with STG.

Its *indications* are: To cover small areas on face to get good cosmetic results, e.g. after excision of basal cell carcinoma.

How to take a split thickness graft?

Instrument used is skin-graft knife (Humby's knife). It has adjustable depth gauge that helps in deciding the depth of STG. Another way to take STG is with mechanical or electrical dermatome. The best donor site is convex surface of limb having thick skin, e.g. thigh.

The procedure is performed under anesthesia. During the procedure, ask the assistant to stretch the donor area. Paint the donor area with a lubricant, e.g. petroleum jelly. Then with to and fro movements of knife, take the graft (Figs 27.7A and B). After taking graft, apply temporary saline pack on donor site for a few minutes to lessen the bleeding. Then apply vaseline gauze followed by thick pad on donor site and do pressure dressing. The re-epithelialization of donor bed occurs in 1-2 weeks.

The procured skin graft is spread over a wooden block lubricated with petroleum jelly. The graft is then fenestrated with a scalpel blade (Fig. 27.8). These fenestrations allow blood and exudates to escape and minimize graft loss due to hematoma formation.

Clean the recipient area and gently apply graft over it taking care that it is not put upside down (Figs 27.9A and B). Skin graft may be anchored with skin sutures. Apply vaseline gauze followed by thick padding and then do pressure dressing. On putting the graft on recipient area, the space between the two is occupied by plasma which makes the graft survive for initial 48 hrs. After that vascular connections develop between recipient area and the graft. So, initial 48 hrs is the critical period when graft can be lost.

The STG is taken up only by areas where recipient bed is vascular (Fig. 27.10). It cannot be taken up by

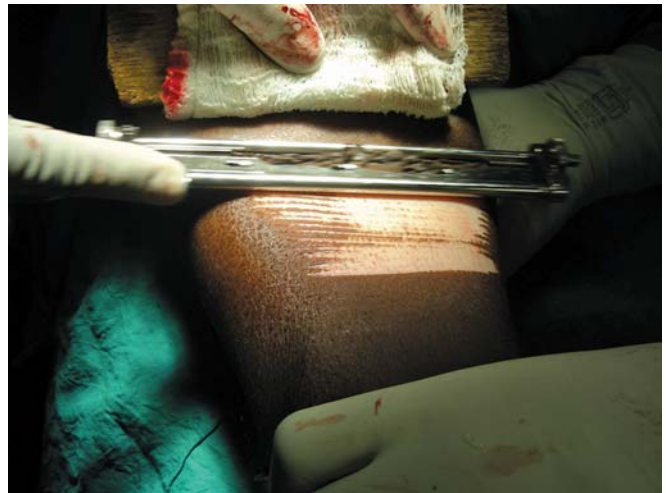


Fig. 27.7A: Split thickness graft being taken from right thigh using Humby's knife



Fig. 27.7B: Split thickness graft being divided with a scissors from donor bed



Fig. 27.8: Harvested split thickness skin graft with multiple fenestrations made by stab incisions

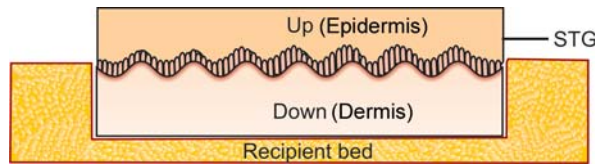


Fig. 27.9A: Placing split thickness graft on recipient bed

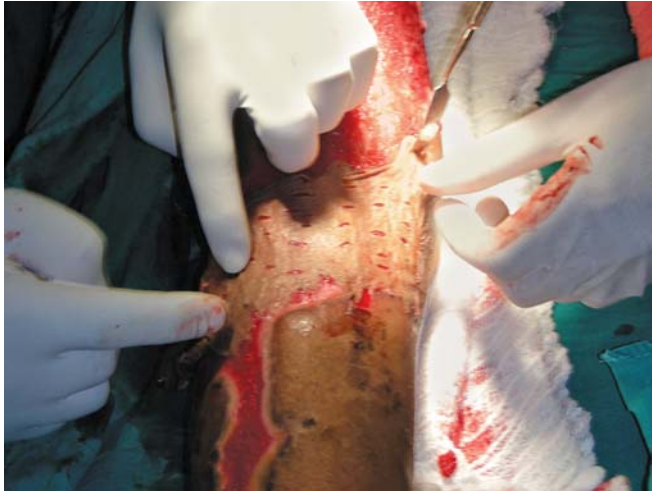


Fig. 27.9B: Skin graft being spread over recipient area

avascular areas, e.g. bone without periosteum, bare cartilage, bare tendons and cavities. The dressing of recipient area is done on 4-5th day.

Causes of Graft Loss

The causes of graft loss are:

- Graft is put up side down and raw area of graft not touching the raw area of wound.
- Hematoma between graft and recipient area.



Fig. 27.10: Skin graft taken up at two weeks

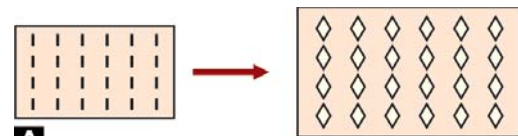
- Infection.
- Improper immobilization.

Mesh Graft

The STG is passed through a mesher so that it enlarges in size on stretching. It helps in covering a large surface area. Moreover fenestrations help in better drainage and prevent graft rejection (Fig. 27.11A).

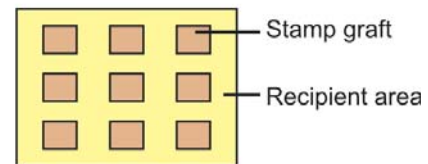
Stamp Graft

Small square pieces of STG are prepared and placed at a distance on raw area (Fig. 27.11B). Intermediate raw areas will heal by spreading of epithelium from small graft. It is useful in large recipient areas when available graft is inadequate. Also if there is any infection or bleeding in the raw area, it will drain out easily and get absorbed in the dressing. However, the cosmetic appearance of grafted area is very poor.



A

Mesh graft before and after stretching



B

Stamp graft put on recipient bed

Figs 27.11A and B: Mesh graft and stamp graft

Contraindications of skin grafting are given in Box 27.7.

Pedicle Skin Flap

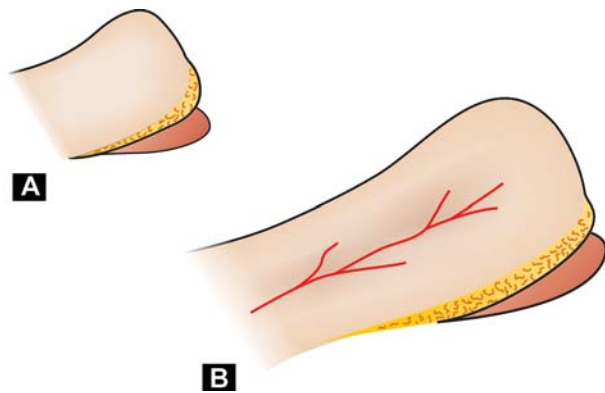
- A skin flap, contrary to free skin graft, retains a continuity by vascular attachment to the donor area.
- Thus, it is a tongue of tissue that consists of skin along with variable amount of underlying structures and is used to cover a defect.
- It is useful in covering the areas having bone without periosteum, bare cartilage, bare tendons and cavities (Box 27.8).
- If a flap is raised from the tissue adjoining the defect, it is called *local flap*.

Box 27.7: Contraindications of skin grafting

- Infection of recipient bed (β hemolytic streptococcal infection is absolute contraindication)
- Unhealthy granulation tissue
- Adjoining infected wound
- Avascular wound (exposed bone, tendon, cartilage, etc.)

Box 27.8: Indications of flap reconstruction

- To cover wound where skin grafting is not possible (exposed bone, cartilage, tendon)
 - To fill wound cavities with tissue loss
 - To cover wound with exposed vital structures (vessels, nerves)
 - To cover orthopedic implants or synthetic mesh
 - To improve cosmetic effect (breast reconstruction following mastectomy)
- If transfer of flap involves moving the tissue at a distance from the primary defect, it is called *distant flap*.
 - Based on **vascular anatomy**, flaps are of two types:
 - Random pattern flap*: In this flap, there is no vascular pedicle supplying the skin flap and the vascular pattern is of random nature (Fig. 27.12A). So, a long flap cannot be raised and there is strict limitation of length to breadth ratio (1:1 or 1:1.5). Example is cross finger flap.
 - Axial pattern flap*: There is definite vascular pedicle in the long axis of the flap. So, a long flap can be raised with less restriction on its width (Fig. 27.12B). Examples are the deltopectoral flap



Figs 27.12A and B: Raising of random pattern flap and axial pattern flap

based on perforating branches of internal mammary artery, forehead flap based on anterior branch of superficial temporal artery.

- Based on their **contents**, flaps are of various types:
 - Fascio cutaneous flap*: It consists of deep fascia along with overlying skin. Inclusion of deep fascia allows greater extension and mobility because blood vessels perforating the subcutaneous tissue are not disturbed, e.g. deltopectoral flap (Fig. 27.13), forehead flap (Fig. 27.14). The deltopectoral flap can be used to cover the defects in lower face and neck.

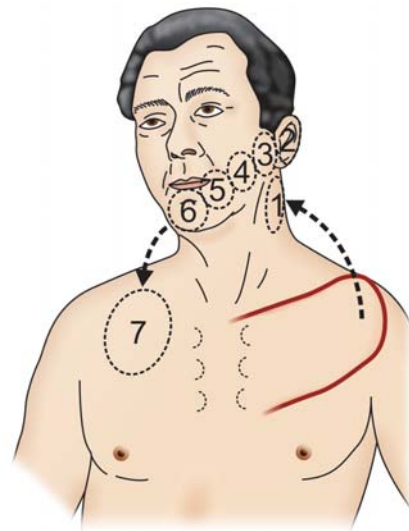


Fig. 27.13: Deltopectoral flap



Fig. 27.14: Forehead flap to cover cheek defect. Donor area is covered with STG

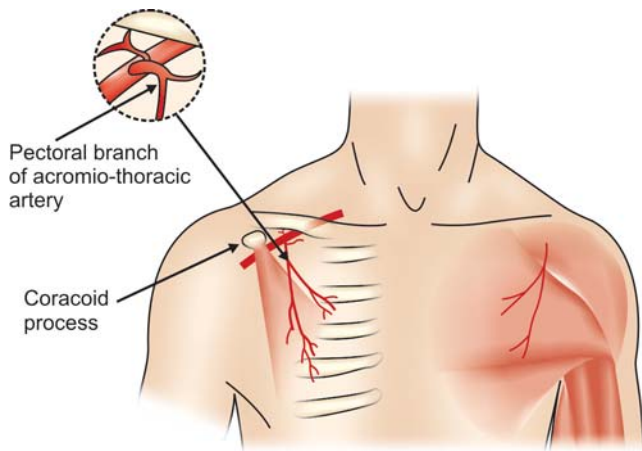


Fig. 27.15: Vascular pedicle of PMMC flap



Fig. 27.16C: RND completed, marking for PMMC flap



Fig. 27.16A: Secondaries neck with skin involvement



Fig. 27.16D: PMMC flap filling gap in neck



Fig. 27.16B: Secondaries neck—incision markings

- b. *Myocutaneous flap*: It consists of muscle along with overlying skin. It is particularly useful in covering bare bone, e.g. pectoralis major flap based on thoracoacromial artery (Fig. 27.15). Figures 27.16A to D show radical neck dissection (RND) being done for secondaries neck involving the skin and resultant defect is being covered with pectoralis major myocutaneous flap (PMMC flap).
- c. *Microvascular free flaps*: A free flap is raised while isolating its neurovascular bundle. This free flap can be taken to any part of the body. The vessels in the recipient site are anastomosed with vessels of free flap using microvascular technique. This technique can help in transferring a piece of bone, nerve, intestine, etc. from one place to another.

28

Chapter

Surgical Suturing

Sanjay Marwah

Suturing is defined as sewing together two structures or tissues using suture threaded on a needle. It can be described under following headings:

- I. Surgical needles.
- II. Suture materials.
- III. Suturing techniques.

SURGICAL NEEDLES

These are sharp pointed instruments used for guiding the thread for suturing or passing a ligature around a vessel.

Parts of a Needle

The parts of a needle are tip, body and eye (Fig. 28.1). The eye is the weakest part of the needle. Hence, needle should never be held near its eye.

Classification

The needles can be *classified* as follows:

On the Basis of its Eye

- Eyeless needles (atraumatic)
- Needles with eye (traumatic)

Eyeless needle has suture swaged to it at the blunt end of the needle. Hence, it causes less trauma. However, it is more costly and can be used only once. It is used in fine surgery, e.g. face, blood vessels, etc.

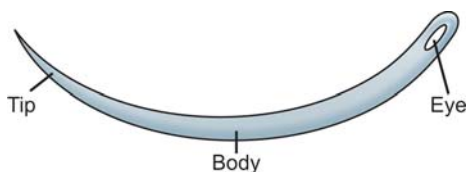


Fig. 28.1: Parts of a needle

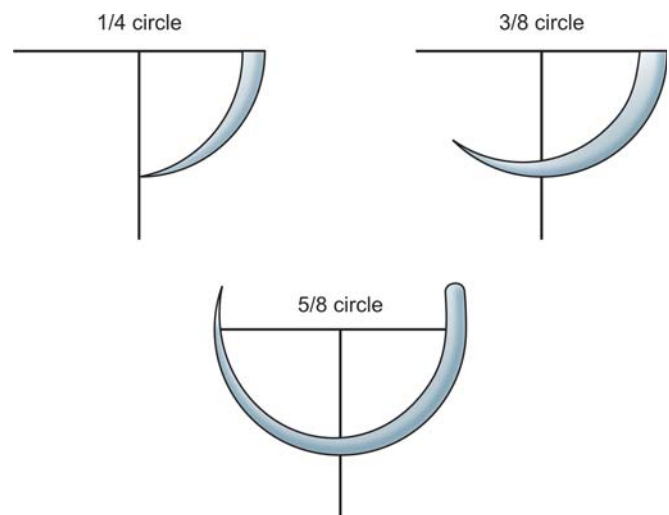


Fig. 28.2: Basic needle curvatures

On the Basis of its Shape

- Straight needle
- Curved needle:
 - i. 1/4 circle
 - ii. 3/8 circle
 - iii. Half circle
 - iv. 5/8 circle (Fig. 28.2)

Straight needles are used for suturing superficial tissues (skin, fascia) without using needle holder. These are rarely used these days.

The curved needles traverse the tissues with circular movement and help in working in the depth. In more confined operative sites, greater curvature needles are required.

On the Basis of its Edge

- Round body needle
- Cutting needle

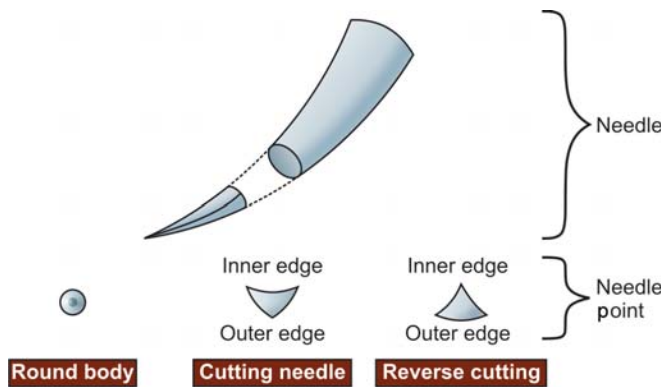


Fig. 28.3: Different types of needle points

- Reverse cutting needle
- Taper cut needle (Fig. 28.3).

Round body needle has a rounded tip that separates the tissue fibers rather than cutting them. It is used in suturing of soft tissues (vessels, intestines).

Cutting needle has two opposing cutting edges on outside and third edge on inside curve of the needle. Thus, its point looks triangular in cross-section. It is used for suturing tough tissues (skin, fascia).

Reverse cutting needle has triangular edge like cutting needle. But the two opposing cutting edges are on inside and third edge on outside curve of the needle. This improves the strength of the needle and increases its resistance to bending in tough tissues (aponeurosis).

Taper cut needle has reverse cutting tip limited to the point of the needle which then tapers out to merge smoothly into round cross-section. Thus, it combines the initial penetration of reverse cutting with minimized trauma of round body needle. This needle is ideally suited for cardiovascular surgery.

Caution: All the needles (and other sharp instruments) should be sterilized by chemical methods (e.g., glutaraldehyde). These should never be boiled or autoclaved as it may damage the sharp edge of the needle.

SUTURE MATERIALS

Classification

The suture materials are classified as follows:

1. *Absorbable:* The sutures are digested by tissue enzymes and removed by phagocytosis. These are of two types:

- A. *Natural*
Catgut
Fascia lata
Kangaroo tendon
Beef tendon.
- B. *Synthetic*
Polyglactin (Vicryl), Polyglycaprone (Monocryl)
Polyglycolic acid (Dexon-S)
Polydioxinone (PDS)

2. *Non-absorbable:* The sutures remain in the body.

These are of three types:

- A. *Natural*
Silk
Cotton
Linen
- B. *Synthetic*
Polyamide
Polyester
Polypropylene
- C. *Metals*
Stainless steel
Silver wire
Titanium

The suture material may be monofilament (prolene) or polyfilament (silk). The polyfilament suture may be twisted or braided.

The advantage of monofilament suture is that it does not allow the bacterial lodgement and thus can be used in presence of infection. However, its disadvantage is that knots slip in monofilament suture due to its plain surface.

The polyfilament suture has good knotting property but the drawback is that it allows bacteria to lodge in its fibers.

Size of Sutures

The size or diameter of suture is based on United States Pharmacopeia (USP). This system used '0' size as baseline. As size decreased below '0', the numbers are added with zero, e.g., 5-0 or 00000 suture is smaller in diameter than 4-0 or 0000 suture. As suture diameter increases above '0', numbers are assigned to the suture size, e.g., 1, 2, 3, 4, are increasing diameters of the suture.

Principles for Selecting Sutures

1. Slow healing tissues like skin and fascia should be sutured with nonabsorbable sutures.

2. Rapidly healing tissues like muscles, intestines should be sutured with absorbable sutures.
3. When cosmetic effect is important, use monofilament, nonabsorbable sutures of small size (e.g. 3-0 prolene). It has minimal tissue reaction. On the other hand, absorbable sutures (e.g. catgut) elicit severe tissue reaction and scar formation.
4. In presence of infection, use monofilament suture as it prevents bacterial lodgment.
5. In malnourished patient, healing is delayed. Hence, use nonabsorbable suture so that tissues are kept in approximation for longer period.
6. For anastomosis of ducts (submandibular duct, parotid duct, common bile duct), always use absorbable sutures. In such situation, nonabsorbable suture will act as a nidus for precipitation of salts leading to stone formation.

Characteristics of Ideal Suture Material

1. Uniform diameter and uniform tensile strength.

2. Adequate tensile strength till tissue healing is complete.
3. It should produce secure knots without cutting or slipping.
4. It should have easy handling.
5. Minimum tissue reaction.
6. It should not allow bacterial lodgment.
7. It should be non-allergic.
8. It should be less expensive and freely available.

Properties of various suture materials are given in Box 28.1 and Box 28.2.

SUTURING TECHNIQUES

Types of Surgical Knots

Various types of surgical knots are:

Reef Knot

It is the most commonly used knot and has the advantage that it does not slip (Fig. 28.4). While tying

Box 28.1: Properties of absorbable sutures

Suture	Source	Tensile strength	Tissue reaction	Remarks
Catgut	Collagen from submucosa of sheep's or cow's intestine	7-10 days	Moderate	Used in rapidly healing tissues (subcutaneous fat)
Chromic catgut	Catgut treated with 20% chromic acid	3-4 weeks	Moderate (less than plane catgut)	Used in slow healing and infected tissues, ligation of vessels.
Polyglactin (vicryl)	Synthetic copolymer of lactide and glycotide	60% at two weeks	Minimal	Excellent tissue handling, can be used in infected tissues. Less tissue reaction. Used in gut anastomosis, ligation of pedicles (thyroid vessels).
Polydioxanone (PDS)	Synthetic (Polyester polymer)	70% at two weeks, 50% at four weeks, absorbed by hydrolysis at 180 days	Minimal	High tensile strength, relatively inert. It glides through tissues due to smooth surface. It is monofilament, so minimal chances of infection. It requires multiple knots (5 or more) for security. It has high cost.
Polyglycaprone (Monocryl)	Synthetic copolymer of glycolite and caproladone	60% at 7 days, 30% at 2 weeks, absorption by hydrolysis at 90-120 days	Minimal	High initial tensile strength, relatively inert, not affected by infection, smooth surface, high cost and require multiple knots. Used for gut anastomosis.

Box 28.2: Properties of non-absorbable sutures

Suture	Source	Tensile strength	Tissue reaction	Remarks
Silk	Silkworm	6 months	Moderate-to-high	Easy handling and knotting, does not become limp or brittle. Avoid in infected tissues. Used in skin sutures, vessel ligation, gut anastomosis.
Cotton	Vegetable origin	50% at 6 months	Minimal	Easy knotting and handling, cheap and freely available. Avoid in infected tissues. Used for skin suturing and vessel ligation.
Stainless steel wire	Alloy of iron, nickel and chromium	Infinite	Minimal	Used for interdental wiring in fracture mandible, bone suture in fracture patella. Poor handling, knot may break, skin discomfort.
Polypropylene (Prolene)	Synthetic polymer of polypropylene	Infinite	Minimal	Resists infection because it is monofilament. Very smooth and glides through tissues. Prolonged tensile strength even in infected areas. Multiple knots required due to poor knotting. Useful in vascular surgery because it is less thrombogenic.
Nylon	Synthetic polyamide polymer	15-20% loss per year	Minimal	Less irritant, cheap, knot is slippery. Infection rate is high due to crevices in braided nylon. Used for skin sutures, tendon repair.

the knot, care should be taken to draw the ends in right direction to keep the knot square.

Granny Knot

It is not secure and is likely to slip. Hence, it should not be used (Fig. 28.4).

Surgeon's Knot

If tissues are approximated forcefully, there is tension on the suture. It is likely to slip after first knot is tied. Hence, first twist is doubled so that it does not slip and it is called as surgeon's knot (Fig. 28.4). It is also useful when thicker suture material is used (e.g. for tying large vessels) or when suture material is slippery (e.g. prolene).

The knots can be tied using hands (Figs 28.5A to F) or instruments (Figs 28.6A to F). For fine suture materials (e.g. used for plastic procedures) instruments are preferred since tying with hands is clumsy and difficult.

Once the sutures are tied, the method of cutting is very important. Non-absorbable skin sutures (e.g. silk) are cut long (1 cm) so that they may be easily identified and removed at a later date. Within body cavities, silk is cut shorter but catgut is cut longer because it tends to swell later. So catgut knot may get undone if thread is short. Similarly prolene thread is cut longer because knot is likely to open due to slippery nature of the thread.

Various **methods of suturing** are as follows:

1. **Continuous suturing:** It is used for closure of long wounds (e.g. rectus sheath closure in laparotomy).

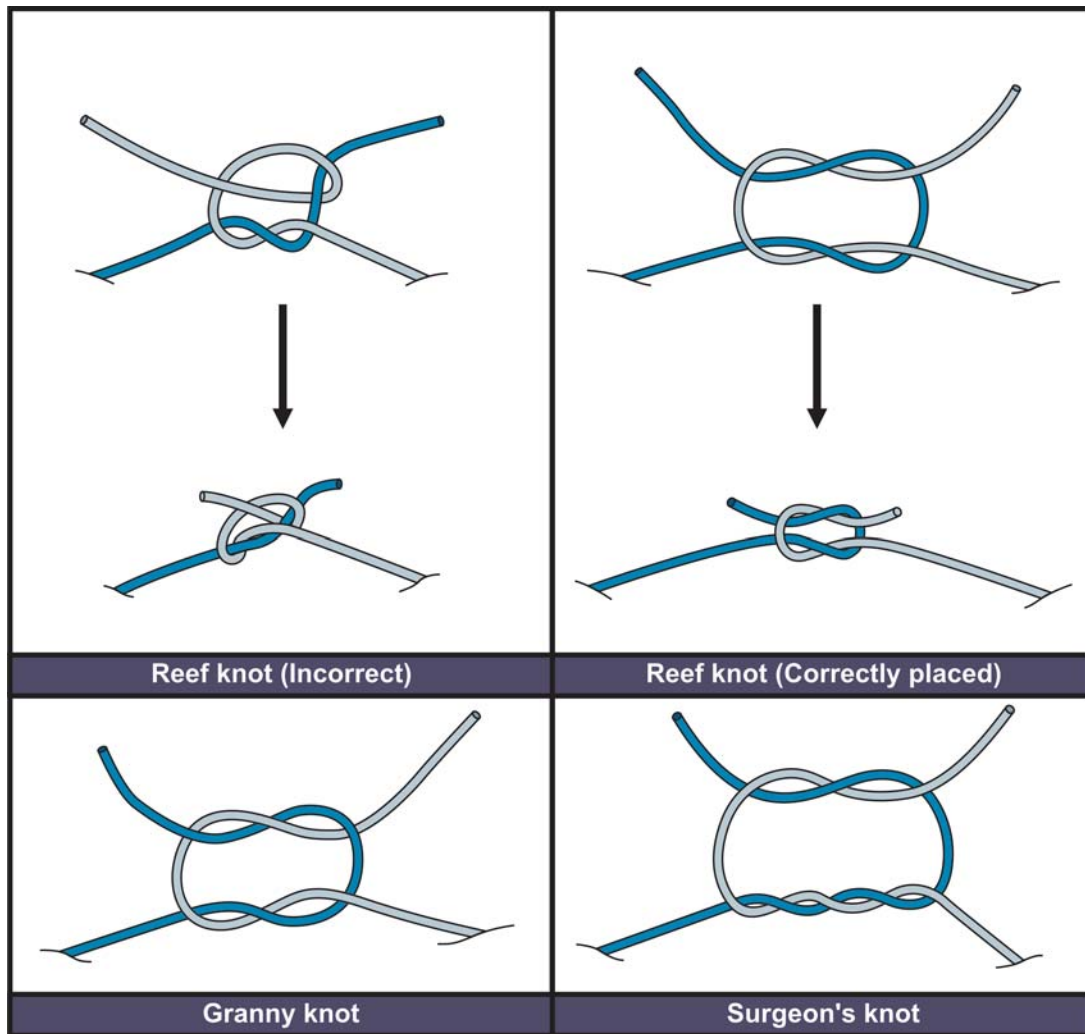


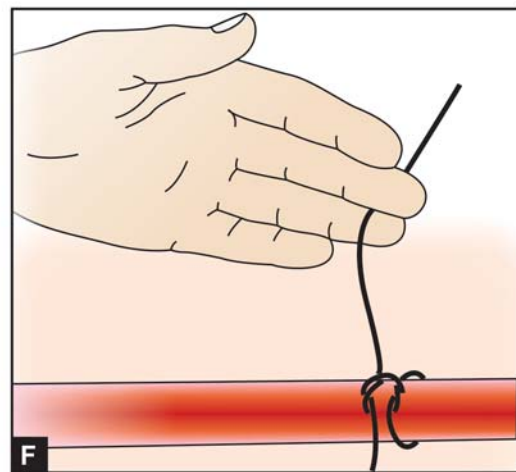
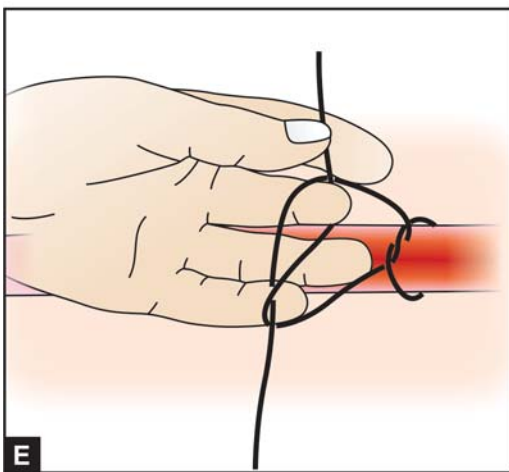
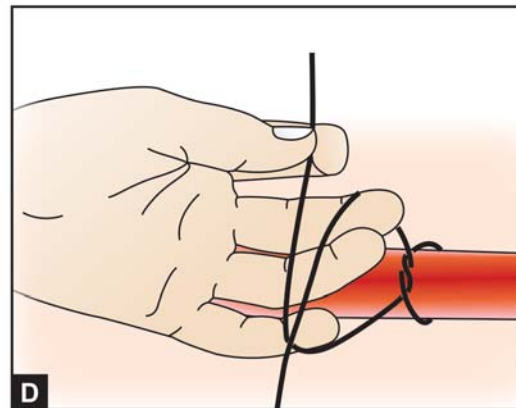
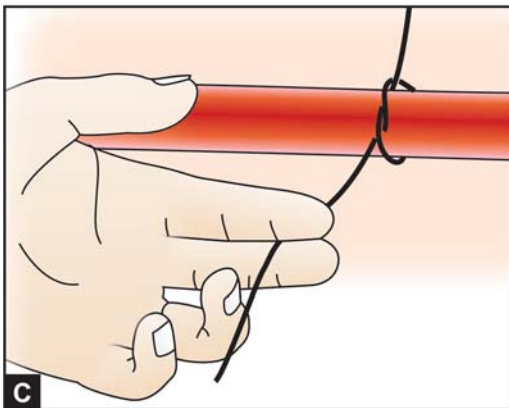
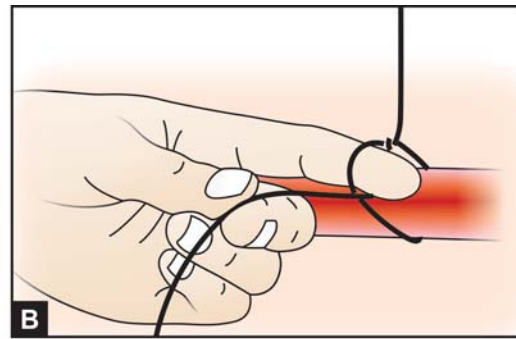
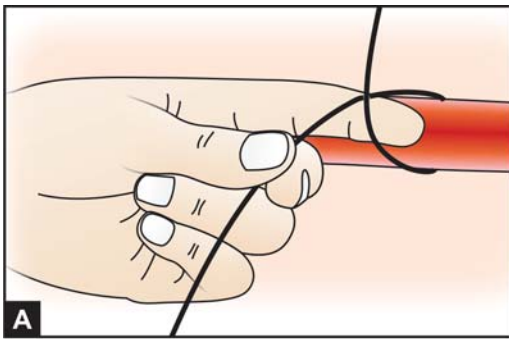
Fig. 28.4: Various types of knots

wound) (Fig. 28.7). Its advantages are that sutures can be applied very rapidly and efficiently. It also has good hemostatic effect as bleeding vessels from the cut edge are compressed with continuous sutures. Its disadvantages are that if suture breaks at one point, the whole wound will gape. In case of hematoma or infection, one cannot remove part of a suture to drain the wound. Moreover, if suture is pulled too tight, the edges may tend to overlap producing purse string effect.

2. **Interrupted suturing:** It is used for closure of skin wounds. Infected areas are also closed with widely spaced interrupted sutures so that pus and exudates can be drained from in between sutures (Fig. 28.8).

Its advantage is that if one or two sutures are removed, the remaining wound does not gape. Its disadvantage is that it is time consuming and requires more suture material.

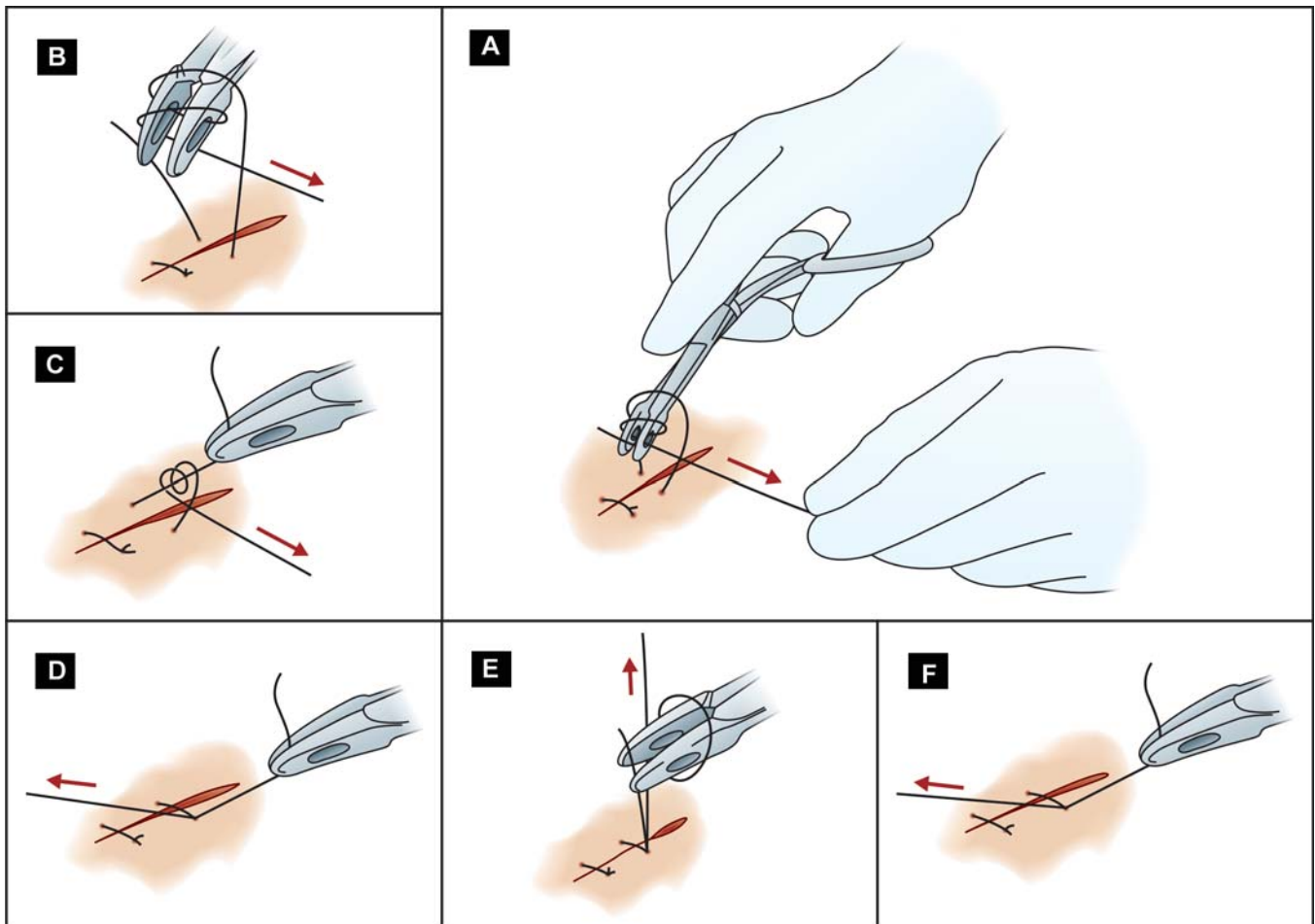
3. **Blanket suturing:** It is a continuous type of suturing with a difference that the needle is passed through loop before pulling each stitch (Fig. 28.9). Its advantage is that it will avoid purse string effect on suture line and tissues will not bunch up.
4. **Mattress suturing:** It is useful in areas where skin is loose and margins are likely to overlap on suturing (e.g. scrotum). It is also used for suturing cut muscles and tendons where simple interrupted sutures are likely to cut through. In this suture, the needle starts



Figs 28.5A to F: Method of tying a reef knot with left hand

from one side, passes to the other side and then comes back to the same side through separate punctures. In second bite, needle comes back taking

edge of the skin incision so that there is no skin overlap on tying the suture (Fig. 28.10). The mattress suture can be applied as horizontal or vertical



Figs 28.6A to F: Instrumental tying of a knot

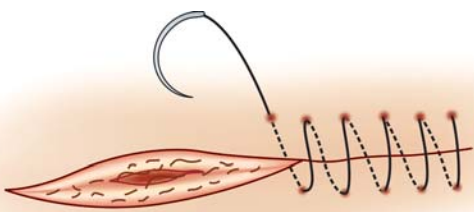


Fig. 28.7: Continuous suturing

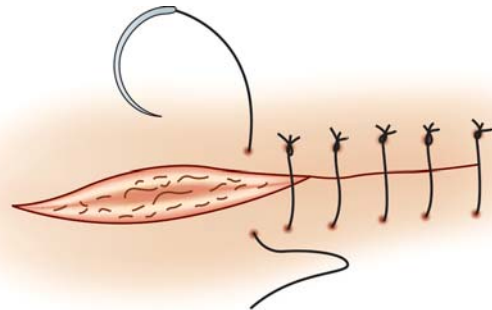


Fig. 28.8: Interrupted suturing

sutures. Horizontal mattress sutures prevent overlap of edges (e.g. skin incision) while vertical mattress sutures prevent cutting through the tissues (e.g. muscle).

5. *Subcuticular suturing:* It is used in areas where cosmetic appearance is important, e.g. face. The needle takes bites through dermis only and continuous suture is passed uniformly at the same

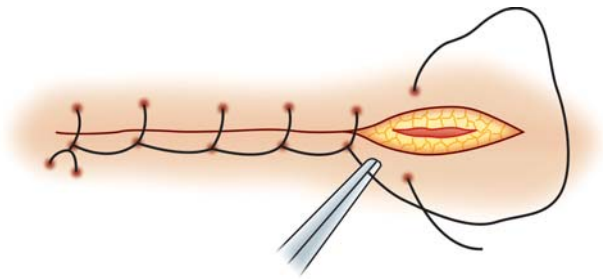


Fig. 28.9: Blanket suturing

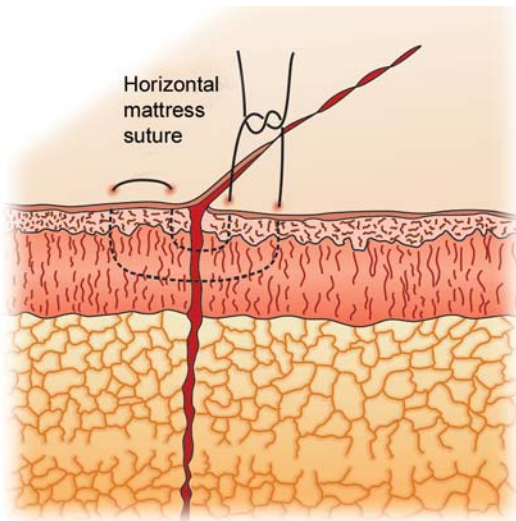


Fig. 28.10: Horizontal mattress suturing

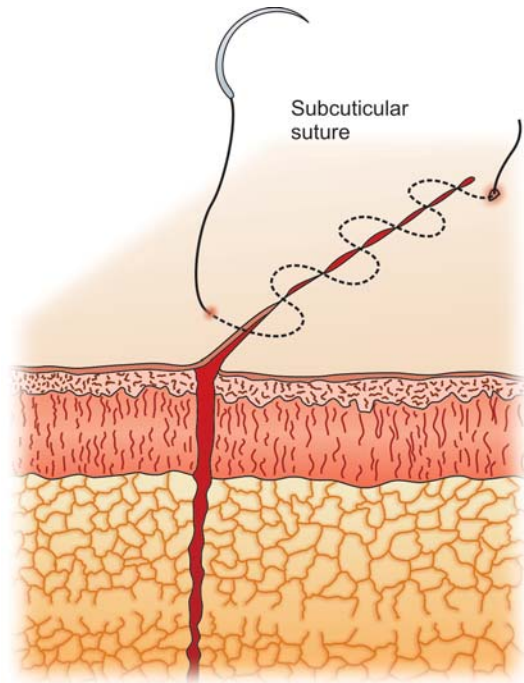


Fig. 28.11: Subcuticular suturing

level without gaps in the linear direction (Fig. 28.11). Its advantage is that cross marks of simple skin sutures are avoided.

29

Chapter

Surgical Instruments

Sanjay Marwah

INSTRUMENTS USED FOR CLEANING AND DRAPING

Cheattle's Forceps (Fig. 29.1)

- It is a large, heavy forceps with curved blades.
- The blades have large serrations that help in getting better hold of sterilized instruments and linen.
- The long handle avoids touching sterilized articles with hands while picking them up.
- The long blades are kept dipped in a bottle containing antiseptic solution (savlon, cidex, etc.).
- It has no lock.
- Its uses are to select and pick autoclaved instruments, drapes, towels, etc.

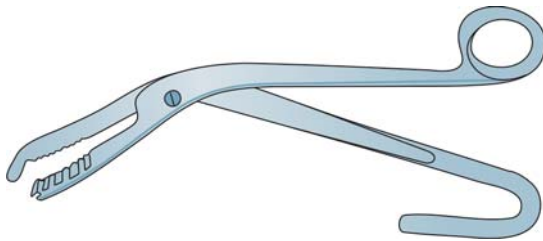


Fig. 29.1: Cheattle's forceps

Rampley's Sponge Holding Forceps (Fig. 29.2)

- It has two long blades with rounded and fenestrated end.
- The inner surface of the end is serrated for better grip.
- It has catch-lock mechanism for holding the sponge firmly.
- Its uses are:
 - To hold the swab used for painting antiseptic solution in the field of operation before starting the surgery.

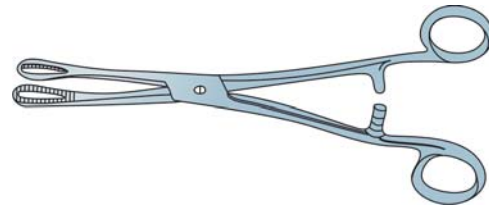


Fig. 29.2: Rampley's sponge holding forceps

- It can be used as a blunt dissector with the swab while dissecting in the depth, e.g. lumbar sympathectomy.
- It can be used to press any oozing in the depth with a swab or packing cavities, e.g. prostatic fossa after prostatectomy.
- It can be used to hold soft hollow organ during dissection, e.g. holding neck of the gallbladder during cholecystectomy.
- It can be used to hold cervix in a pregnant patient.

Mayo's Towel Clip (Fig. 29.3)

- It is a light but strong instrument having small and curved blades.
- The tips of the blades are sharp and pointed for better grip of linen.
- It has catch-lock mechanism for better grasp.

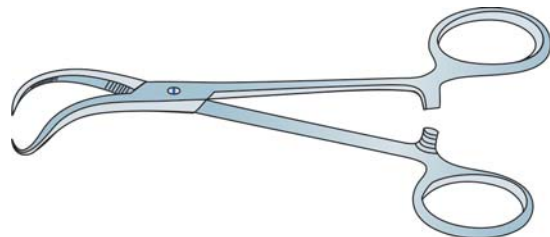


Fig. 29.3: Mayo's towel clip

- Its uses are:
 - ☐ To fix the drapes for proper exposure of operative field.
 - ☐ To fix the cautery lead and suction tube so that these do not fall off the operation table during handling.
 - ☐ It can be used in place of tongue holding forceps but it bites the tongue.
 - ☐ It can be used to hold the ribs while elevating and fixing the fractured ribs.

Moynihan's Tetra-towel Clip (Fig. 29.4)

- It is similar to Mayo's towel clip except that it is longer and has four teeth instead of two.
- Its uses are:
 - ☐ To fix the drapes.
 - ☐ To cover the cut margins of incision with towels from all sides so as to minimize the chances of wound contamination.

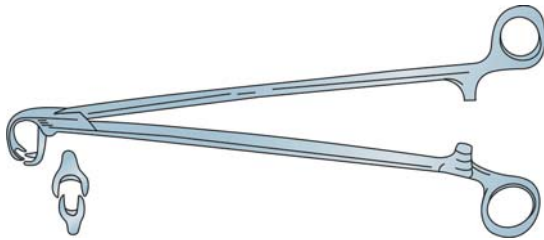


Fig. 29.4: Moynihan's tetra-towel clip

DISSECTING FORCEPS

- The forceps is so designed that it is normally open and on applying pressure on the grip, there is very precise closure of the tip.
- The outer surface is grooved to give firm grip.
- There are two main types based on the tips:

Toothed Forceps (Fig. 29.5)

It has toothed tip that interlocks on closure. The toothed tip gives a firm and better hold over the tissues. It is used to hold tough tissues like fascia, skin, etc.



Fig. 29.5: Toothed forceps

However, the presence of teeth at the tip makes it traumatizing forceps.

Non-toothed or Plain Forceps (Fig. 29.6)

It has serrations at the tip that exerts grip over a wide area and is non-traumatizing forceps. It is used to hold soft tissues like muscles, intestines, etc.



Fig. 29.6: Plain forceps

TISSUE HOLDING FORCEPS

It is used to hold the tissues firmly for the purpose of dissection or tissue apposition at the time of suturing.

Various examples of tissue forceps are:

Allis Tissue Forceps (Fig. 29.7)

- It is traumatizing type of tissue forceps.
- Its tip has got triangular expansion with sharp teeth that interlock on closing.
- It has got a catch-lock mechanism (Spencer Wells) that locks it on closure.
- It is used to hold tough structures like skin, fascia, aponeurosis, etc.

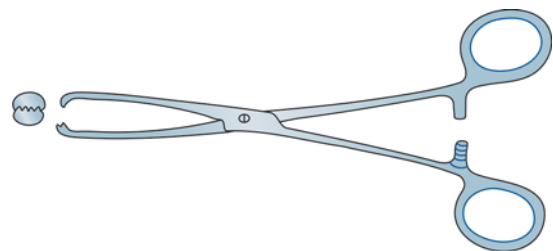


Fig. 29.7: Allis tissue forceps

Babcock's Tissue Forceps (Fig. 29.8)

- It is non-traumatizing type of tissue forceps.
- Its tip has got triangular expansion without any teeth and transverse serrations on inner aspect.
- It has got a catch-lock mechanism for locking.
- Its blades are fenestrated that makes the instrument light weight. The fenestrations also allow soft tissues to budge through them.

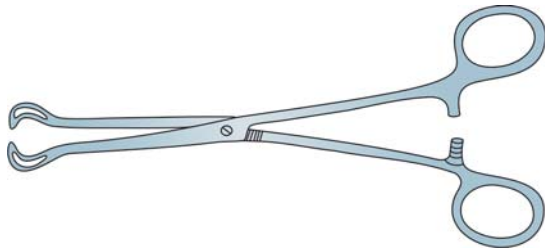


Fig. 29.8: Babcock's tissue forceps

- It is used to hold soft and delicate tissues like thyroid gland, lymph nodes, muscles, intestines, appendix, etc.

Lane's Tissue Forceps (Fig. 29.9)

- Its function is in between Allis forceps and Babcock's forceps and can be used for holding both tough as well as delicate tissues.
- Its tip is thicker and expanded with a bigger opening than Babcock's forceps.
- The tip of the blades has got a single tooth.
- It has got a catch-lock mechanism for locking.
- Its uses are:
 - ☐ To hold tough tissues like skin, fascia, etc.
 - ☐ To hold soft tissues like lymph node for biopsy.
 - ☐ The appendix is held between blades and not by the tip.
 - ☐ Sometimes it can be used in place of towel clip for fixing the towels during draping.

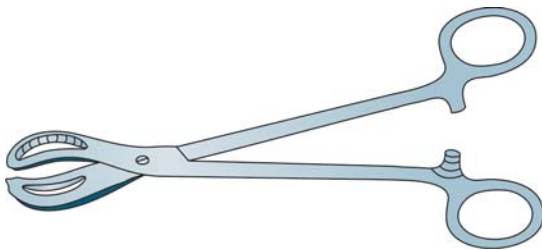


Fig. 29.9: Lane's tissue forceps

Tongue Forceps (Fig. 29.10)

- It has a single spike at the tip that holds the tongue at one point and does not allow slipping of the tongue.
- The catch-lock mechanism provides firm grip.
- Its use are:
 - ☐ To stop hemorrhage from the tongue.

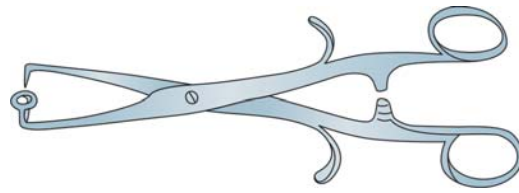


Fig. 29.10: Tongue forceps

- ☐ To hold the tongue during any surgery of the tongue.
- ☐ To prevent falling back of tongue in an unconscious patient.

FORCEPS USED FOR HEMOSTASIS

The most important method to control bleeding during surgery is to apply an arterial clamp on bleeding vessel. It stops bleeding by apposing the cut ends of the vessel. The techniques of hemostasis are described in Chapter 20: Principles of Operative Surgery, Diathermy and Radiotherapy.

Artery Forceps (Hemostat)

It has following features:

- The inner margins of the blades are serrated and on closure, the blades are tightly apposed without any gap in between.
- The blades are held together by means of a catch-lock mechanism.
- The blades are conical and blunt. Based on the shape of the blades, artery forceps can be straight or curved.
- The artery forceps has following types:

Small or Mosquito Forceps (Fig. 29.11)

It is very small in size and has relatively pointed tips.

- It is used for holding small bleeding points.
- It is very useful in plastic surgical procedures, e.g. cleft lip, cleft palate.
- Straight mosquito forceps is used for:
 - ☐ holding 'stay sutures'.
 - ☐ holding gauze pallets for blunt dissection (Peanut).

Medium Artery Forceps

It is the most commonly used type of artery forceps. It can be straight (Fig. 29.12) or curved artery forceps (Fig. 29.13).

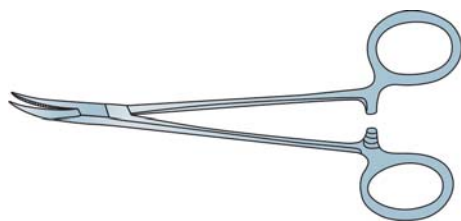


Fig. 29.11: Small or mosquito forceps

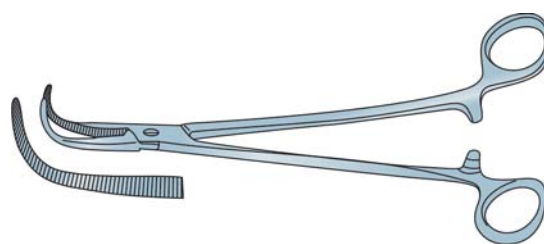


Fig. 29.14: Lahey's forceps

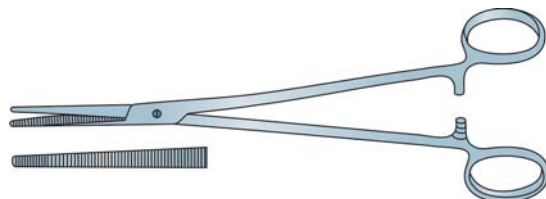


Fig. 29.12: Straight artery forceps (Medium)

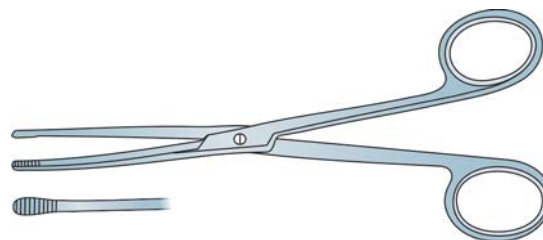


Fig. 29.15: Sinus forceps

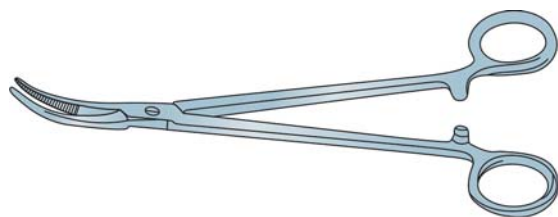


Fig. 29.13: Curved artery forceps (Medium)

It has following uses:

- To control bleeding.
- To crush base of appendix during appendicectomy.
- To hold free ends of 'stay sutures'.
- To hold the tape of abdominal packs to prevent any chance of these packs being forgotten inside abdominal cavity.
- To hold cut edge of fascia, peritoneum, aponeurosis.
- To break the loculi of abscess cavity.
- To hold needle for suturing if needle holder is not available.

Large Artery Forceps

It has long blades and is used for holding vessels in the depth, e.g. renal vessels, cystic artery during cholecystectomy.

Lahey's forceps (Fig. 29.14) is an example of large artery forceps with right angle at the operating end. It is very useful in dissection as well as ligation of major vascular pedicles, e.g. superior thyroid pedicle during thyroidectomy.

Sinus forceps (Fig. 29.15) is like an artery forceps except that it has no catch-lock mechanism. It has serrations confined to the tip of the blades so as to hold the wall of abscess cavity, e.g. for biopsy. In Hilton's method of abscess drainage, after giving skin incision, sinus forceps is thrust to open the abscess cavity. The catch-lock mechanism is not provided to prevent accidental crushing of underlying vital structures, e.g. vessels and nerves.

Kocher's Forceps (Fig. 29.16)

- It is a toothed variety of hemostatic forceps.
- It has a single sharp tooth at its tip that is meant for a better grip.
- It has serrations on blades and catch-lock mechanism similar to artery forceps.
- It is available in curved and straight form.

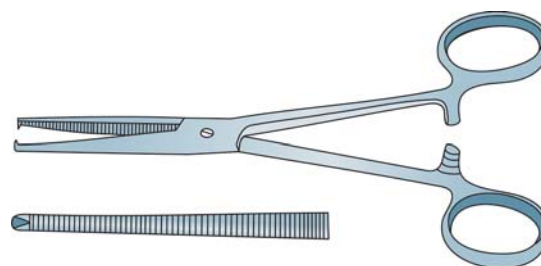


Fig. 29.16: Kocher's forceps

- Its uses are:
 - ☐ To hold thyroid vessels (original use of Kocher's forceps).
 - ☐ To hold strap muscles of neck during thyroidec-tomy before dividing them.
 - ☐ To hold the retracting cut ends of the vessels in tough fibrous tissues like scalp, soles and palms.
 - ☐ To hold gauze, pallets for blunt dissection.
 - ☐ To hold rib during rib resection.
 - ☐ For artificial rupture of gestational membranes during delivery.

Pott's Bulldog Clamp (Fig. 29.17)

- It is a small paper clip like instrument.
- It has strong jaws with serrations on inner margins. The jaws may be covered with rubber tubing to prevent crushing of vessels.
- It has spring loaded handle that ensures a secure grip of the vessels.
- It is used to clamp large vessels during surgery on these vessels. Also, in case of accidental bleeding from the large vessels, the vessels can be immediately clamped to stop bleeding, e.g. internal jugular vein.

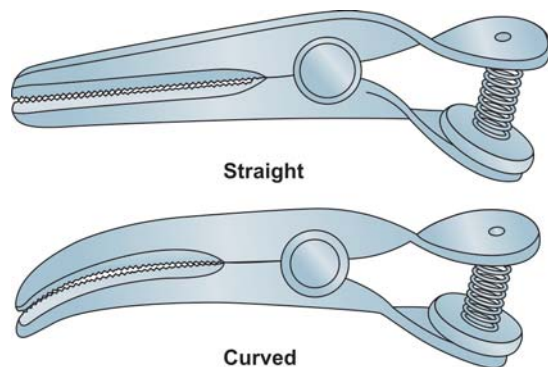


Fig. 29.17: Pott's bulldog clamp

Well's Arterial Clamp (Fig. 29.18)

- It is a long heavy instrument meant for hemostasis.
- It has double right-angled jaws to hold large vessels.
- The jaws have longitudinal serrations.
- It is used as a pedicle clamp for splenectomy, nephrectomy.

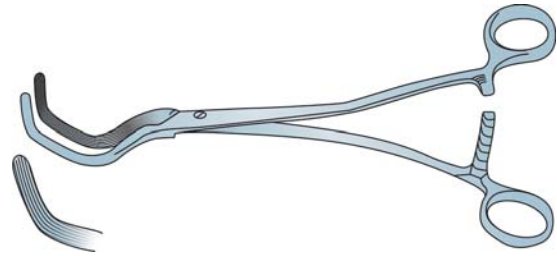


Fig. 29.18: Well's arterial clamp

SUTURING INSTRUMENTS

Surgical Needles

See chapter 28—Surgical Suturing.

Aneurysm Needle (Fig. 29.19)

- It is so called because it was primarily invented to ligate the aneurysmal dilation of arteries.
- It is hook-shaped instrument with blunt tip. The blunt tip avoids injury to the vessels.
- There is an 'eye' at the tip through which a suture is threaded. The tip of the needle is then passed behind the vessel and suture is pulled and tied to ligate the vessel.
- Its uses are:
 - ☐ To ligate an aneurysm.
 - ☐ During venesection, silk suture is hooked and passed around the vein to ligate it.
 - ☐ It can be used to ligate any vessel in continuity.



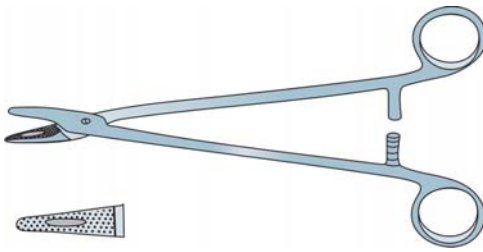
Fig. 29.19: Aneurysm needle

Needle Holder (Fig. 29.20)

- It is used to hold the curved needle while suturing.
- The blades are serrated and have catch-lock mechanism like an artery forceps.
- The differences between needle holder and artery forceps are given in Box 29.1.
- The blades are smaller and have crisscross serrations with a groove for holding the needle.

Box 29.1: Differences between needle holder and artery forceps

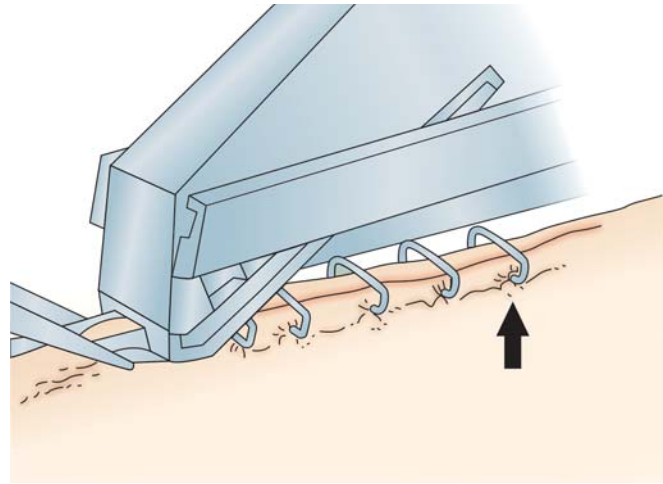
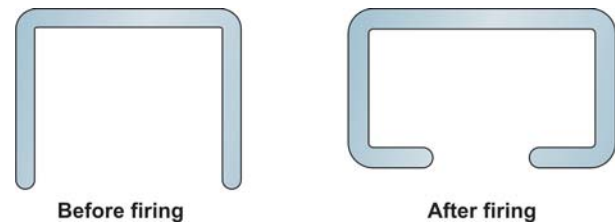
Needle holder	Artery forceps
Jaws very small in comparison to shaft.	Jaws are almost half the length of shaft.
Crisscross serrations on inner surface of jaws (See Fig. 29.20).	Transverse serrations on inner surface of jaws (See Fig. 29.12).
Longitudinal groove is present on serrated surface for holding the needle.	No groove on serrated surface.
Jaws are blunt and stout.	The jaws are blunt, conical and not very heavy.

**Fig. 29.20: Needle holder**

- It may be straight or curved. The straight type is used for surface suturing. The curved type is used for suturing in depth so that tissues are visible while taking bite.
- For suturing, the needle is held with needle holder at right angles at the junction of anterior 2/3rd and posterior 1/3rd of the needle. If needle is held in the middle, then its enough length is not available to pass through the tissues smoothly. If needle is held near its posterior end, it may break as eye of the needle is weakest point. While passing the needle through the tissues, force is applied along curvilinear axis of the needle for easy and smooth movement of the needle. After passing the suture, the knots can be tied with the help of needle holder.

Clip Applicator (Michel's)

- Titanium clips are used for wound closure with help of clip applicator.
- For skin stapling, disposable skin stapler (clip applicator) is available that has preloaded bracket shaped clips.
- The skin margins are approximated with tooth forceps and the skin stapler is applied over the everted skin margins. The stapler is fired by squeezing that forces the clip to enter the skin edges and to assume a final rectangular shape (Figs 29.21A

**Fig. 29.21A: Application of skin staples****Fig. 29.21B: Open and closed shapes of the clips**

- and B). This rectangular shape resists rotation, prevents skin inversion and allows easy removal of the clips.
- Its advantages are that it is simple, easy to use, quick to apply and minimizes tissue trauma. It gives cosmetically clean scar because there is no risk of epithelial downgrowth since there is no track going down through epidermis (unlike needle puncture). It is very useful in closing head and neck incisions, e.g. thyroidectomy incision.
- Its disadvantages are that it is costly and requires special kit for application and removal. It is also not

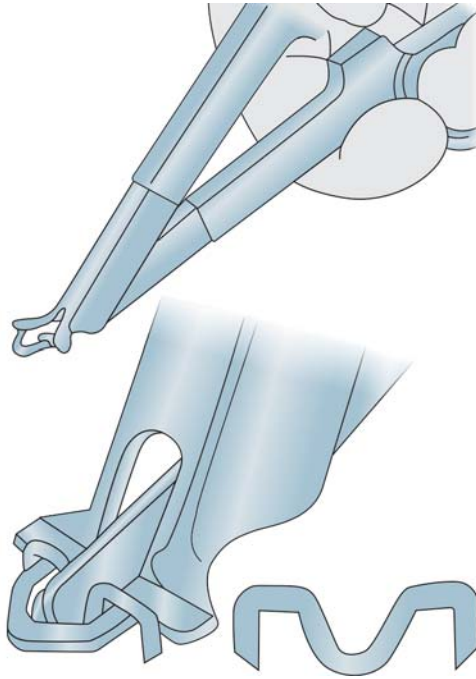


Fig. 29.22: Clip extractor

hemostatic. These days, clips are also used for ligating vessels in depth during open as well as laparoscopic surgeries.

Clips Extractor

- It is a ring-handled instrument that looks like a stitch cutting scissors.
- It has an angled flange on each side of the lower jaw that forces the staple to reform (from rectangular to bracket shape) without twisting in the skin.
- For clip removal, slide the lower jaw of the clip extractor beneath the staple applied in the skin. On squeezing the handle of the extractor, the clip will reform and come out of the skin (Fig. 29.22).

INSTRUMENTS USED FOR MAKING INCISION

Bard Parker Knife Handle

It has serrations for better grip (see Fig. 20.2A).

Detachable Blades

These are of different size and shapes. Details are given in chapter 20: Principles of Operative Surgery, Diathermy and Radiotherapy.

SCISSORS

It is the instrument used for blunt as well as sharp dissection and cutting various structures and sutures.

It can be of various shapes and sizes:

- Curved or straight
- Sharp or blunt pointed.

Straight Scissors

Straight scissors with sharp points is used for cutting excess length of sutures or for suture removal (Fig. 29.23).

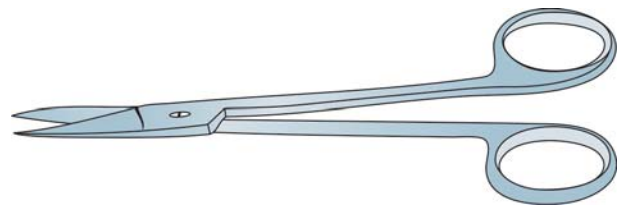


Fig. 29.23: Straight scissors with sharp points

Straight scissors with blunt points is used for cutting bandages and gauze (Fig. 29.24). It can also be used for cutting excess length of suture material once suture has been tied.

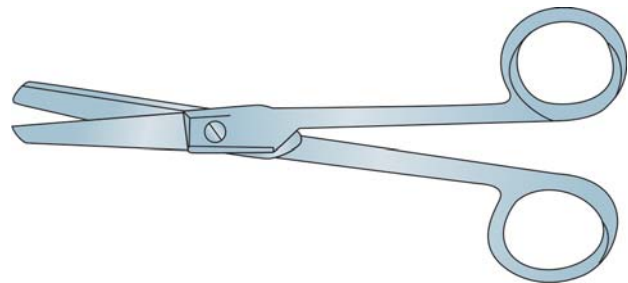


Fig. 29.24: Straight scissors with blunt points

Curved Scissors (Fig. 29.25)

It is also called dissecting or Mayo's scissors.

It is used to dissect tissue planes and to divide important structures.

The curvature is useful in dissection at a depth.

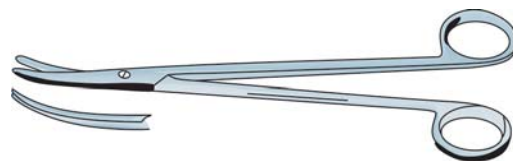


Fig. 29.25: Curved scissors

RETRACTORS

These are the instruments used to retract the tissues for better exposure of surgical field.

Various types of retractors are:

Nerve Hook/Retractor (Fig. 29.26)

- It is a small delicate instrument with a blunt hook at its distal end.
- It is used to lift nerves and retract nerves during dissection.



Fig. 29.26: Nerve hook

Skin Hook (Fig. 29.27)

- It is a small delicate instrument with a sharp hook at its distal end.
- It is used to retract skin flaps, e.g. during thyroidectomy.
- It is also used at the time of closure of skin flaps. Two skin hooks are applied one at each of the skin incision so as to approximate the skin edges.
- It is also used to retract the skin edge while applying subcuticular stitches.



Fig. 29.27: Skin hook

Cat's Paw Retractor (Fig. 29.28)

- It is multi-hooked retractor with pointed edges.
- It is used to retract tough structures like scalp skin, fascia of palms and soles.

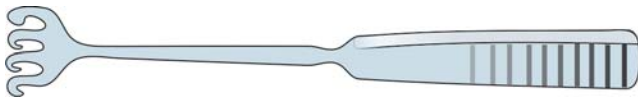


Fig. 29.28: Cat's paw retractor

Kocher's Thyroid Retractor (Fig. 29.29)

- It is a self-retaining type of retractor that has two flanges connected by a lockable joint.
- Each flange has multiple pointed hooks meant for retracting skin flaps.

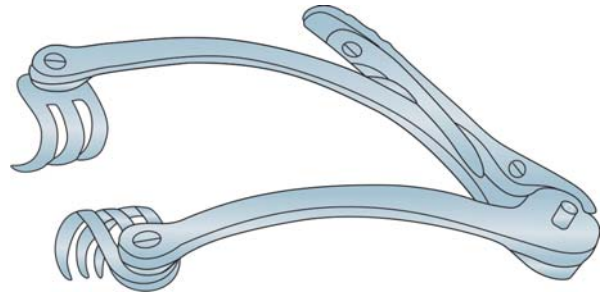


Fig. 29.29: Kocher's thyroid retractor

Joll's Thyroid Retractor (Fig. 29.30)

- It is also a self-retaining type of retractor that has two flanges connected by an adjustable screw mechanism.
- Each flange has a locking clip meant for holding the skin flaps.

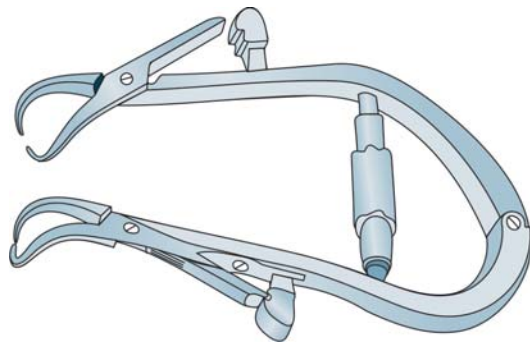


Fig. 29.30: Joll's thyroid retractor

Doyen's Mouth Gag (Fig. 29.31)

- It is a self-retaining retractor having catch-lock mechanism.
- The blades are semicircular and bent to hold the jaws well.

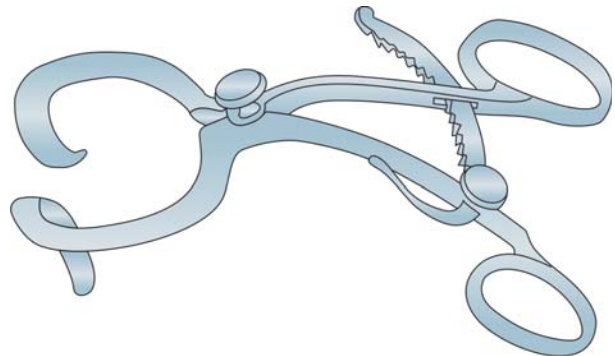


Fig. 29.31: Doyen's mouth gag

- It is used:
 - ☐ To keep the mouth wide open during operations inside the mouth on tongue, cheek, tonsil, etc.
 - ☐ To open the mouth in unconscious patient for oral toilet.
 - ☐ To aid movement of temporo-mandibular joint in fibrous or false ankylosis.

Langenbeck's Retractor (Fig. 29.32)

- It has a single blade at right angle to a long handle.
- The handle is fenestrated to make it light weight.
- It is a superficial retractor used to retract layers of abdominal wall during laparotomy.

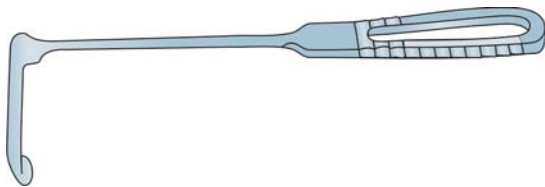


Fig. 29.32: Langenbeck's retractor

Czyrny's Retractor (Fig. 29.33)

- It is a retractor with small blade on one end and biflanged hook on the other end.
- The hook and blades are directed in opposite direction.
- The handle is fenestrated to make it light weight.
- It is also used for superficial retraction of layers of abdominal wall during laparotomy.
- The biflanged end helps in tissue exposure while applying the last deep stitch during wound closure.

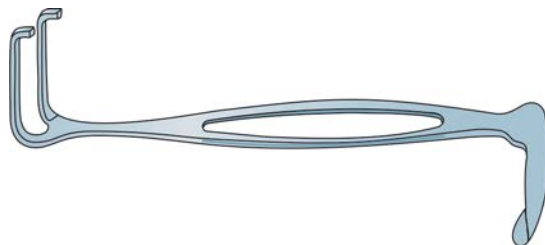


Fig. 29.33: Czyrny's retractor

Morris Retractor (Fig. 29.34)

- It is a large, strong retractor with anteroposteriorly curved blades.
- The concavity of the blade gives wider space for working.

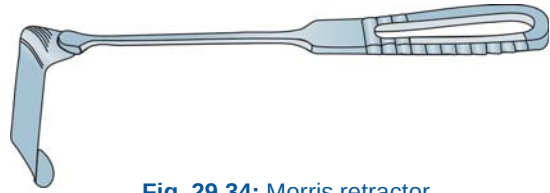


Fig. 29.34: Morris retractor

- The shaft of the instrument is fenestrated to make it light weight.
- It is used to give deep retraction by retracting strong structures like abdominal wall muscular, e.g. exposure for kidney.

Deaver's Retractor (Fig. 29.35)

- It is a large retractor with a broad and gently curved blade.
- It is used for retracting deep intra-abdominal viscera, e.g. liver, spleen, urinary bladder, uterus, etc.

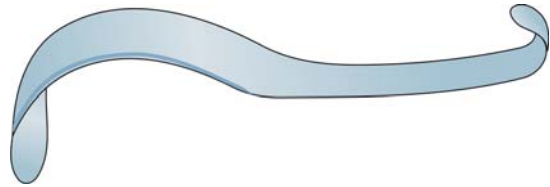


Fig. 29.35: Deaver's retractor

Doyen's Retractor (Fig. 29.36)

- It is a heavy retractor with rounded blade.
- It is used to retract abdominal wall after the peritoneum is opened during laparotomy.
- The rounded edge prevents the peritoneum from slipping under the instrument.

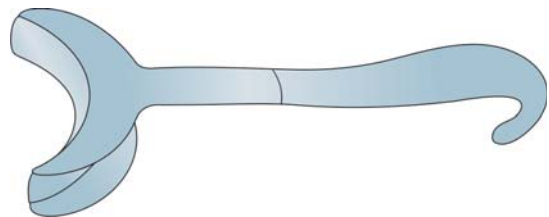


Fig. 29.36: Doyen's retractor

Self-retaining Abdominal Wound Retractor (Fig. 29.37)

- It is a heavy retractor with three adjustable blades.
- Two lateral blades are like Doyen's retractors used for retracting abdominal wall on two sides.

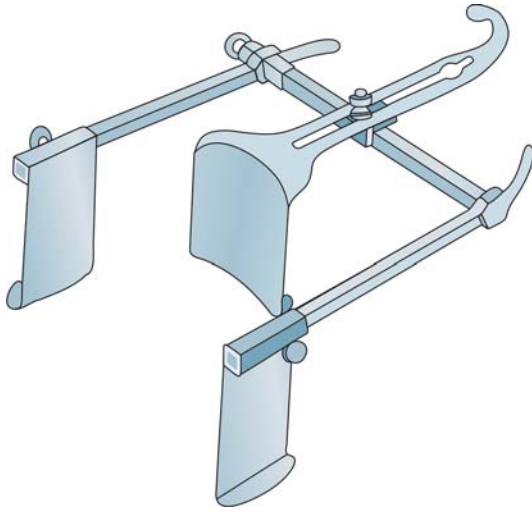


Fig. 29.37: Self-retaining abdominal wound retractor

- Third blade is like Deaver's retractor used to retract pelvic structures at lower end of the laparotomy incision.
- The position of the blades can be adjusted by means of screw locks.
- It is used in long abdominal surgeries and decreases the number of assistants required for retraction.

MISCELLANEOUS INSTRUMENTS

Trocar and Cannula (Fig. 29.38)

- It has two parts, outer hollow metallic tube (cannula) and inner solid metallic pointed rod (trocar).
- It is used
 - ☐ To drain hydrocele fluid.
 - ☐ To drain empyema gallbladder.
 - ☐ To drain superficial liver abscess.

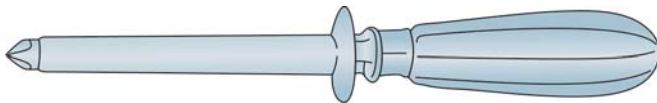


Fig. 29.38: Trocar and cannula

Hudson's Brace and the Burr (Fig. 29.39)

- It is a heavy instrument with a brace and the burr (drill).
- There is a set of drills with rounded as well as pointed tips that can be locked with brace.
- It is used to make an opening in the vault of skull for gaining access to intracranial structures.

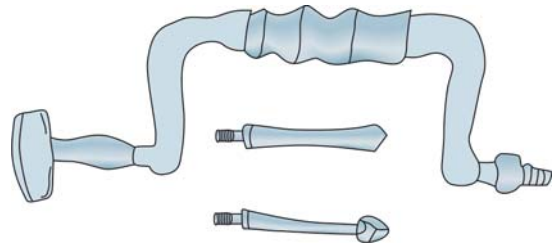


Fig. 29.39: Hudson's brace and the burr

- In emergency, it is used for draining extradural or subdural hematoma.

Periosteal Elevator (Fig. 29.40)

- It is an instrument used to lift the periosteum before cutting a bone, e.g. mandible, rib, etc.
- It has following parts:
 - ☐ Handle that is flat and grooved.
 - ☐ Thumb rest that is oval in shape with corrugated surface to give a firm grip.
 - ☐ Cutting edge that is bevelled only on one side.
- After exposing the bone, periosteum is first incised with a knife and then stripped with a periosteal elevator to denude the underlying bone.
- With thumb placed on thumb rest of the periosteal elevator, fingers grasp the handle and bevelled edge helps in elevating the periosteum with sliding movements.



Fig. 29.40: Periosteal elevator

Gigli Saw (Fig. 29.41)

- It is an instrument used to cut a bone (osteotomy).
- It has multiple braided steel wires hooked with two handles.
- The wire is passed behind a bone and moved to and fro with the help of two handles.
- The braided wire provides rough surface that enables the saw to cut sharply through hard bone.
- During to and fro movement of the wire, constant dripping of saline on the wire helps in its smooth movements.
- It leads to cutting of the bone without damaging adjoining soft tissues.

- It is useful in dividing bone lying in deep cavity surrounded by important structures.
- It is useful in following operations:
 - ☐ Hemimandibulectomy.
 - ☐ In brain surgery, it is used for cutting the bone between burr holes to raise osteoplastic flaps of vault.
 - ☐ McMurray's osteotomy.

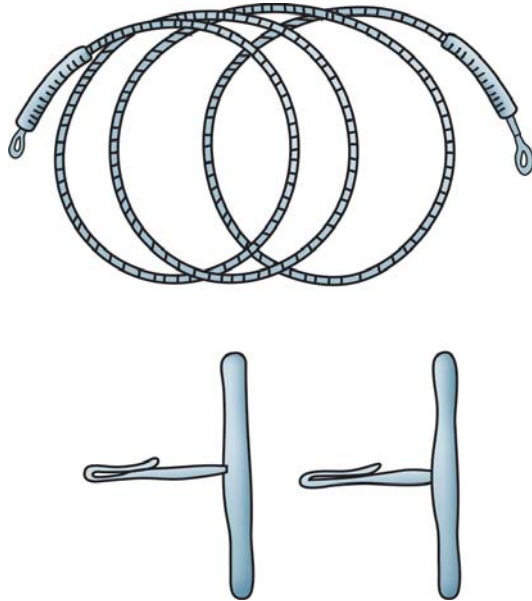


Fig. 29.41: Gigli saw with handles

Bone Nibbler (Fig. 29.42)

- It is a strong instrument with cupped blades having sharp edges.
- The cupped blades are used for nibbling small pieces of bones to smoothen the bone surface.
- The handles have single or double action lever to make it stronger.
- It is available in various sizes and can be straight or curved.
- Its uses are:
 - ☐ To take a bone biopsy.
 - ☐ To enlarge a burr hole in craniotomy.
 - ☐ To enlarge an opening in a bony cavity, e.g. saucerization in chronic osteomyelitis.

Malleable Probe (Fig. 29.43)

- It is a simple instrument that is blunt at one end and has an eye on the other end.

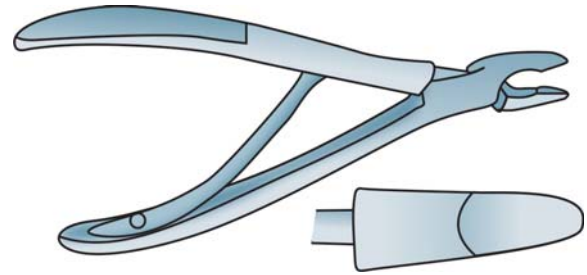


Fig. 29.42: Bone nibbler



Fig. 29.43: Malleable probe

- It is used to explore the tract of sinus and fistula.
- The eye can be used to pass a 'Seton' (a loop of thread) in high fistula.

Probe with Dissector (Fig. 29.44)

Its one end is probe-pointed and the other end is flattened and carries a groove. The pointed end is used as a probe and flattened end is used as a blunt dissector.



Fig. 29.44: Probe with dissector

Brodie's Fistula Director (Fig. 29.45)

- It is a thin long instrument with winged end.
- Its tip is blunt and malleable and can be used for probing the fistula track.
- Its winged end is used for dividing the tongue tie. After opening mouth, tongue is lifted up. The winged end is passed below the tongue and the frenulum is caught in the slit portion. The frenulum is divided with a scissors and an adrenaline pack is put for hemostasis.

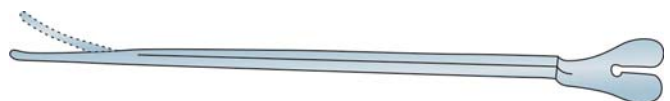


Fig. 29.45: Brodie's fistula director

Kocher's Thyroid Dissector (Fig. 29.46)

- This instrument resembles a scalpel in shape but its edge is blunt.
- The anterior surface of the blunt end has grooves to facilitate dissection.
- The handle has serrations for better grip.
- It is used for blunt dissection of fascial coverings during thyroidectomy.



Fig. 29.46: Kocher's thyroid dissector

Volkman's Scoop (Curette) (Fig. 29.47)

- It is a long instrument with scoops at both ends in opposite directions.
- The sharp edge of the scoop helps in curetting the granulation tissue that collects in the cavity of the scoop.
- The collected granulation tissue can be sent for histopathology as well as for culture and sensitivity.
- It is used:
 - ☐ To curette chronic ulcers and sinuses.
 - ☐ To curette bony cavities (chronic osteomyelitis, osteoclastoma, bone cyst).

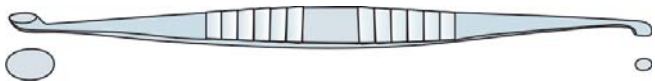


Fig. 29.47: Volkman's scoop (curette)

Humby Skin Grafting Knife (Fig. 29.48)

- The instrument has a handle and a long sheath made of stainless steel.
- While using, a disposable blade can be attached over it.
- There is an adjustable roller on the sheath that adjusts the thickness of the skin graft to be taken.

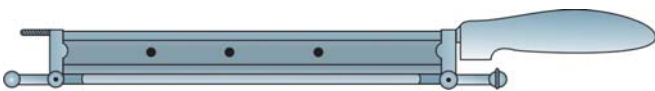


Fig. 29.48: Humby skin grafting knife

- It is used for taking split skin graft. The technique is described in chapter 27: Burns and Skin Grafting.

Myer's Vein Stripper (Fig. 29.49)

- It is a long flexible wire with detachable heads. It looks like an 'accelerator wire' of a scooter.
- It is used for stripping varicose veins in the lower limb.
- The technique of stripping is described in chapter 19: Diseases of Venous System.

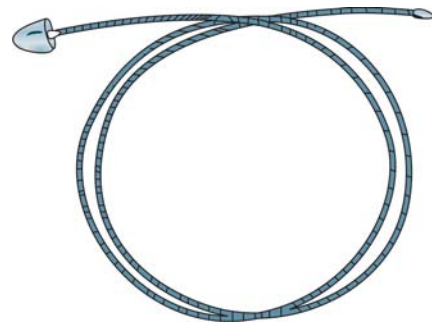


Fig. 29.49: Myer's vein stripper

TRACHEOSTOMY INSTRUMENTS**Cricoid Hook (Fig. 29.50)**

- It has a broad and long handle with a hook at the operating end.
- During tracheostomy, it is to 'hook up' the cricoid cartilage so as to stabilize the trachea.
- Once trachea is stabilized, it can be properly incised without slipping.



Fig. 29.50: Cricoid hook

Tracheal Dilator (Fig. 29.51)

- This instrument has two blunt and curved blades at the operating end.
- There is no catch lock mechanism in the handle.
- The special feature of this instrument is that when handle is closed, the blades at operating end open and vice versa.

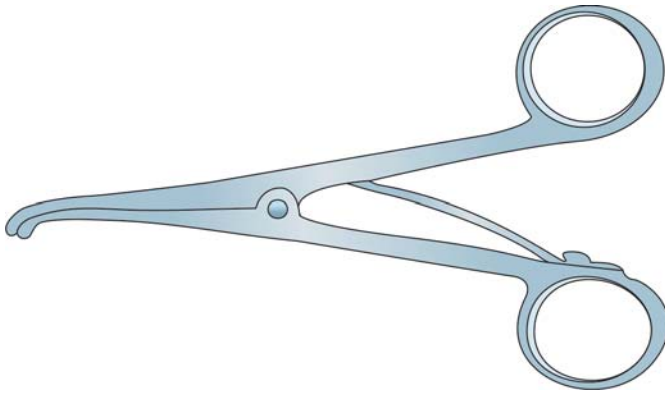


Fig. 29.51: Tracheal dilator

- During tracheostomy, after incising the anterior wall of trachea, tracheal dilator is introduced to open the tracheal wound for putting in tracheostomy tube.

Tracheostomy Tubes

These are made up of two materials—metallic and plastic.

Metallic Tube (Fig. 29.52)

It consists of two components outer biflanged tube and inner tube. There is no cuff. The inner tube is always longer than the outer tube so that outer tube is never blocked by secretions. If inner tube is blocked, it can be removed for cleaning while leaving patent outer tube in place. The wings of outer tube are used for fixing it in position with ribbon tapes tied around the neck.

Plastic Tubes

Modern tracheostomy tubes are made of PVC. These are softer, less irritant, pre-sterilized and disposable tubes. They are available with or without inflatable cuff (Fig. 29.53). Unlike metallic tube, it is used as a single tube. After introducing it in trachea, the cuff is inflated

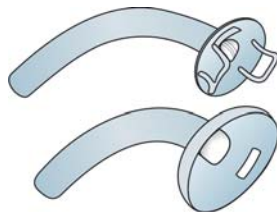


Fig. 29.52: Metallic tubes

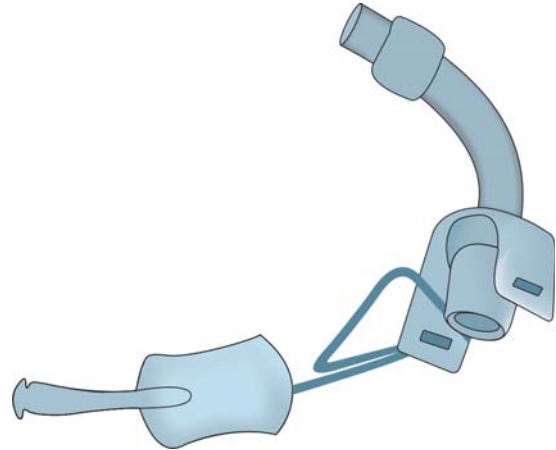


Fig. 29.53: Cuffed plastic tube

with 3-5 ml of air. It prevents leakage of air during mechanical ventilation. It also prevents risk of aspiration pneumonia. In case it is blocked, it needs to be removed for cleaning. While it is being cleaned, the tracheal wound is kept open with a tracheal dilator.

DRAINS

Types of Drain

Corrugated Drain (Fig. 29.54)

- It is made of red rubber or PVC.
- It has corrugations on both surfaces that prevent closure of the wound and allow fluid to drain out freely.
- It is attached to the skin by an anchoring stitch.
- It is used after thyroidectomy, superficial parotidectomy, drainage of an abscess cavity.
- Its drawback is that it is an open drain leading to wound soakage that requires repeated dressings. Also there is risk of infection from outside.



Fig. 29.54: Corrugated drain

Tube Drain

- It is made of red rubber or PVC and has multiple side holes near its tip for free drainage (Fig. 29.55).

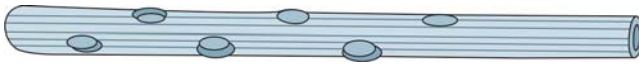


Fig. 29.55: Tube drain with multiple side holes

- It is put in the most dependent part of the wound and taken out through a separate stab incision using the shortest possible route.
- It is attached to a drainage bag for fluid collection. If drainage bag has a suction mechanism, it acts as a negative suction drain (Fig. 29.56).

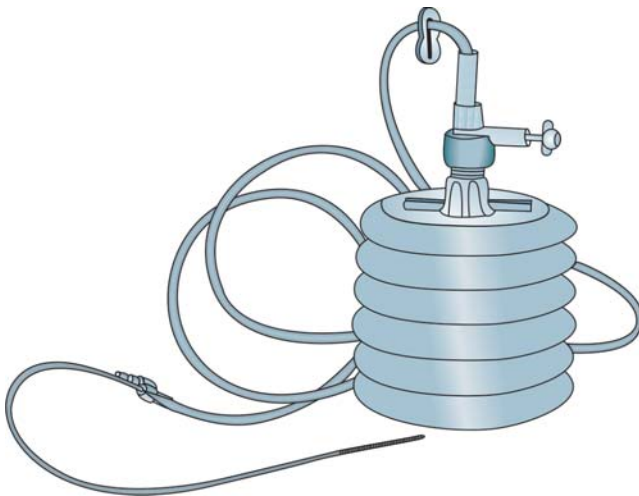


Fig. 29.56: Negative suction closed drainage system

- It is put:
 - ☐ In the neck after thyroidectomy (see Fig. 20.5).
 - ☐ On the face after superficial parotidectomy.
 - ☐ In abdominal cavity after laparotomy.
- Because it is a closed system, there is no soakage and repeated dressings are not required. Exact amount of drainage fluid can be measured. There is minimal chance of infection from outside.
- The drawback is that holes may be blocked by blood clots, debris or surrounding tissues.

General Principles for Putting a Drain

- Drain should be put if there is a potential space in which there are chances of collection after surgery.
- Drain should always be put in most dependent part.
- Drain should always be taken out through shortest and straight route.
- Drain should always be taken out through separate stab wound away from the main incision line. It is to avoid infection of the main wound.

- Drain should be fixed to the skin using an anchoring suture.
- In case of tube drain, patency should be checked regularly.
- Drain is usually removed in 48-72 hrs after surgery unless there is persistent significant discharge.

PLASTIC AND RUBBER INSTRUMENTS

Ryle's Tube (Fig. 29.57)

It is a transparent tube made of PVC (polyvinyl chloride) and measures one meter. The tip of the tube is blunt with lead shots inside it. The lead shots at the tip help passing down of the tube and make it radiopaque. There is also a green colored radiopaque lining along the length of the tube. Thus, on plain X-ray film, exact position of the tube can be assessed.

There are multiple side holes near the tip of the tube that allow suction of gastrointestinal secretions. There are three circular marks on the tube that tell location of the tip inside the stomach (Box 29.2).

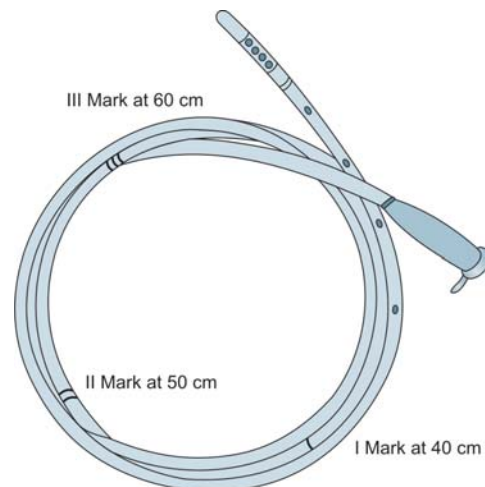


Fig. 29.57: Ryle's tube

Box 29.2: Marks indicating location of the tip of Ryle's tube

Marks of Ryle's tube	Length passed	Location of the tip
First mark	40 cm	Gastroesophageal junction
Second mark	50 cm	Body of the stomach
Third mark	60 cm	Pylorus of the stomach

Uses of Ryle's Tube

- Nasogastric aspiration in acute intestinal obstruction, peritonitis and following bowel surgery.
- In case of gastric hemorrhage, for gastric aspiration and gastric lavage.
- For feeding purpose:
 - ☐ Following faciomaxillary injury.
 - ☐ In unconscious patient (following head injury)
 - ☐ In debilitated patients who cannot take orally.

How to Pass a Ryle's Tube?

Position of the patient: Lying down with extended neck or sitting position.

The tip of the Ryle's tube is lubricated with xylocaine jelly and introduced through the wider of the two nostrils. The tube is pushed gently along the floor of the nose. Once the tube reaches the pharynx, there is reflex coughing and gag. Reassure the patient and ask him to swallow the tube. Giving patient a sip of water helps in sliding down the tube. If tube accidentally enters the trachea, there is violent coughing and flushing of face. The tip of the tube should be immediately withdrawn and the procedure is restarted.

Once the tube has passed correctly into the stomach, its position needs to be checked by one of following methods:

- Inject 2-3 cc of air into the tube with a syringe and auscultate for gurgling sound in the epigastrium.
- Tube aspiration with a syringe shows free aspiration of gastric contents.

Once position of the tube is checked, its outer end is taped to the forehead of the patient. The outer opening of tube is connected to a drainage system for escape of gastrointestinal contents or the opening may be plugged and aspirated intermittently every 3-4 hrs.

Infant Feeding Tube (Fig. 29.58)

It is similar to Ryle's tube except it is much smaller in size and has no lead shot.

It is used in infants and small children for the same purpose as Ryle's tube is used in adults.

It can also be used in place of venesection cannula for venous cut down.

Venesection Cannula (Fig. 29.59A)

It is thin PVC tube available pre-sterile in a double plastic packing. There is a terminal opening with blunt end.

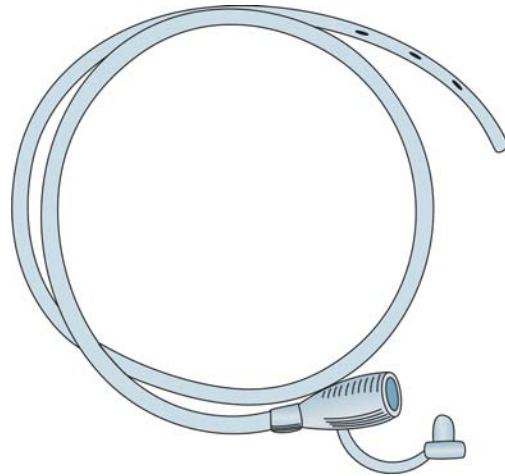


Fig. 29.58: Infant feeding tube

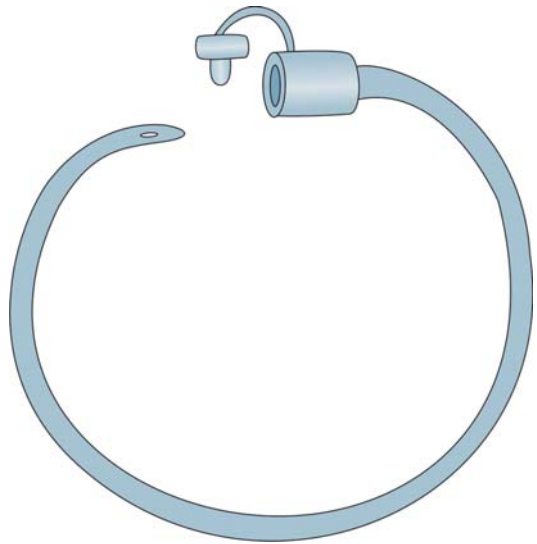


Fig. 29.59A: Venesection cannula

The other end is broad and can be connected to intravenous infusion set. Also there is a cap attached to the broad end that can be used to seal the tube once intravenous line is not in use.

Indications of Venous Cut Down

- Patient in shock requiring rapid intravenous infusion, e.g. burns, septicemia, hemorrhagic shock.
- When intravenous infusion is required for very long time.
- To put CVP line for central venous pressure monitoring.

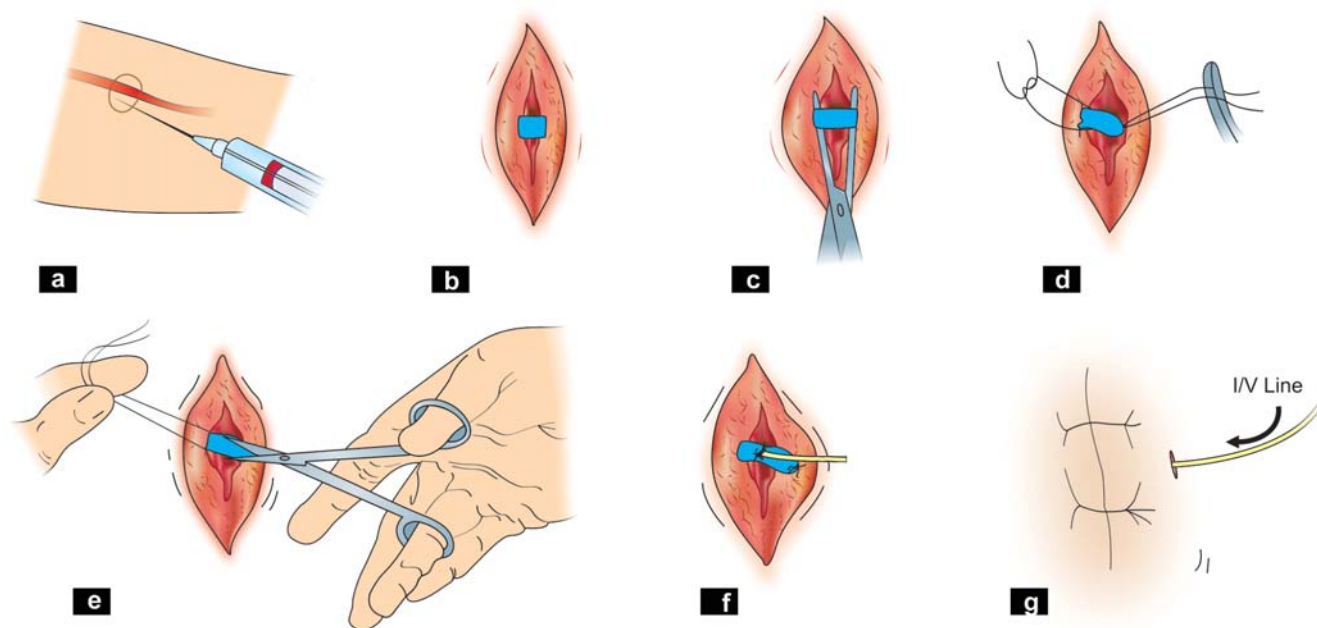


Fig. 29.59B: Steps of venesection

How to do Venesection? (Fig. 29.59B)

The sites of venesection are:

- Long saphenous vein at medial malleolus.
- Cephalic vein in lower arm.
- Femoral vein in the groin.
- Cephalic vein in the deltopectoral groove.

After cleaning and draping the part, a local anesthesia is infiltrated at site of incision. A skin incision is given across the selected vein. The vein is dissected and two threads are passed deep to the vein, one each at either end of the incision. The distal thread is tied off. Thus, vein is ligated and it prevents profuse bleeding on opening the vein.

The vein is transfixing with a needle and a surgical blade is used to cut over the transfixing needle to open the vein. Another way is to use a fine sharp-pointed scissors to make an opening in the anterior wall of the vein.

A venesection cannula is fed into the vein towards proximal limb for 10-12 cm. Free back flow of blood into the cannula tells that it is lying in correct position. The second loop of thread around vein at proximal end of incision is now tied so as to fix the cannula. Now a small skin incision is made distal to the first incision and a subcutaneous track is made between the two incisions.

The free end of the cannula is brought out through this tract and attached to intravenous line. The skin is closed at both sites using interrupted silk sutures and wound is dressed. The cannula should be removed in 3-5 days time to prevent thrombophlebitis.

Urethral Catheters

These are meant for urethral catheterization so as to drain urine from the urinary bladder.

Types

These are of two types:

- Plain catheter—K-90 catheter.
- Self-retaining (indwelling catheter)—Foley's catheter, Gibbon's catheter.

K-90 catheter (Fig. 29.60): It is a plain catheter made of PVC. Its terminal end is blunt and rounded. It has a side hole near the tip. It has the advantage that it is relatively rigid due to plastic make and can negotiate narrow urethra (in urethral stricture, prostatic enlargement). It is used for single catheterization only and it is removed once bladder has been emptied.

Foley's catheter (Fig. 29.61): It is relatively soft and made of latex material. It is self-retaining catheter and

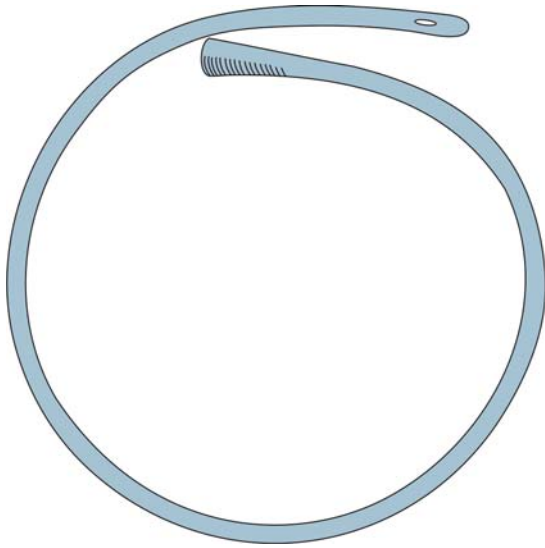


Fig. 29.60: K-90 catheter

can be left in place for a few days. Its lumen consists of a main channel for urinary drainage and a side channel connected to a bulb near its tip. Once the bulb is inflated with water, the catheter is held within internal urinary meatus. The capacity of the bulb is variable (5-30 ml) and is usually mentioned on the catheter. The Foley's catheter is available in various sizes marked by number in French scale.

Catheter diameter in mm = Catheter number/3.

In general, for simple drainage in case of adult urinary retention, 14 F catheter should be used.

Foley's catheter is available as triple lumen catheter as well. The three channels are meant for—urinary



Fig. 29.61: Foley's catheter

drainage, bulb inflation and bladder irrigation (e.g. in case of clot retention).

- *Advantage* of Foley's catheter is that it is self-retaining and is extremely valuable in females.
- *Disadvantage* is that it is semi-rigid and may fail to negotiate narrow urethra. Moreover, draining lumen is relatively small due to presence of second channel for bulb inflation.

Gibbon's catheter (Fig. 29.62): It is also self-retaining catheter made of PVC. It comes in different sizes for males and females as length of urethra in two sexes is different. The catheter has a plastic stellate that makes it stiff and helps in negotiating narrow urethra. It has two plastic ribbons (Gibbon with a ribbon) attached to its upper end. After catheterization, these ribbons are fixed to the genitalia with leukoplast. This catheter is used less commonly these days.

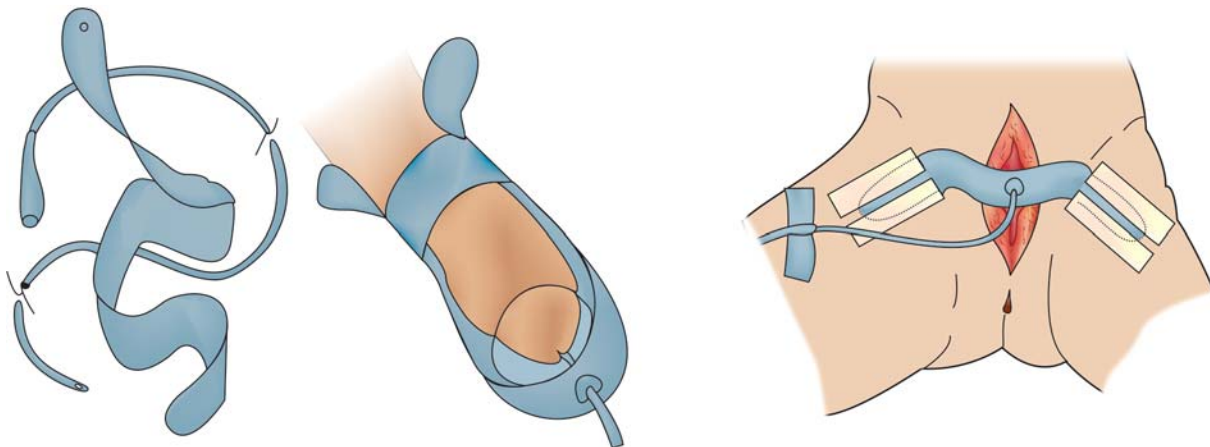


Fig. 29.62: Gibbon's catheter

How to do Urethral Catheterization?

Indications of catheterization

- Acute retention of urine, e.g. postoperative.
- Chronic retention of urine.
- Urinary incontinence, e.g. paraplegia, head injury.
- In patients of shock (septicemic, hypovolemic) for monitoring urine output.
- In operations involving prostate and urinary bladder.
- In patients of pelvic trauma.

Technique of Catheterization

The catheterization should be done with “no touch” technique to prevent infection.

The genitalia (penis or labia) is cleaned with antiseptic solution (povidone iodine). In males, glans penis should be carefully cleaned after retracting the foreskin and wiping the accumulated smegma. Ten cc. of xylocaine jelly is injected into the urethra via external meatus so as to lubricate and anesthetize the anterior urethra. After injecting the lubricant, it should be massaged in the urethra while clamping the glans penis. Now tip of the Foley’s catheter is lubricated and introduced into the urethra with right hand. The shaft of the penis is kept erect by holding with left hand so that catheter passes in smoothly. Once the tip of catheter crosses the bladder neck, urine starts flowing out. The tip of the catheter should be pushed further in so as to avoid inflation of the bulb in the urethra. Then bulb of the catheter is inflated with saline to make it self-retaining. The draining channel is now connected to urinary bag. The bag is hooked on the side of the bed so that it does not fall on the floor and remains below the level of urinary bladder. At the end of the procedure, always remember to replace the foreskin over the glans penis otherwise paraphimosis may develop. The steps of male catheterization are shown in Figures 29.63A to F.

For removal of Foley’s catheter, the bulb is deflated by sucking water through its channel and catheter is pulled out.

Complications of Catheterization

- Urethral injury.
- False passage formation.
- Accidental inflation of bulb in urethra.
- Paraphimosis.



Fig. 29.63A: Xylocaine jelly injected into urethra after retracting prepuce and cleaning glans penis



Fig. 29.63B: Lubricated catheter tip is introduced into external urinary meatus while keeping penile shaft erect



Fig. 29.63C: Whole length of catheter introduced into urethra



Fig. 29.63D: Bulb of the catheter inflated with saline



Fig. 29.63F: Draining channel of the catheter connected to urinary bag; note urine flowing into the bag

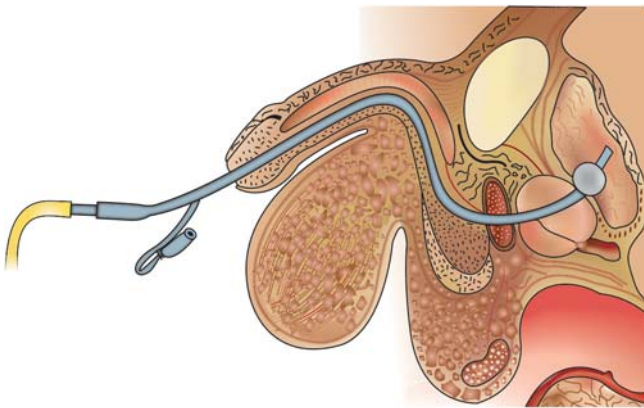


Fig. 29.63E: Inflated balloon of Foley's catheter at bladder neck

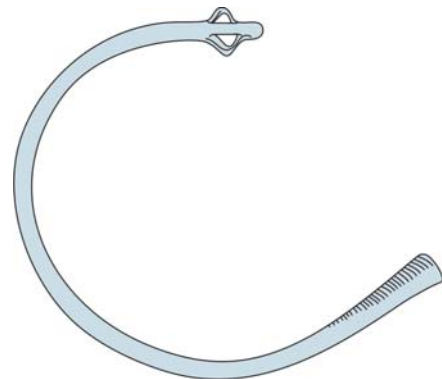


Fig. 29.64: Malecot's catheter

- Urinary infection.
- Retained bulb of Foley's catheter.

Malecot's Catheter (Fig. 29.64)

It is self-retaining catheter made of red rubber. The tip of the catheter is bulbous and winged. The tip is flexible and can be stretched over an artery forceps to facilitate its introduction into a cavity. Once artery forceps is

removed, the bulb is restored at the tip and makes it self-retaining. It uses are:

- For suprapubic drainage of urine by suprapubic cystostomy in case of failed catheterization.
- For closed drainage of any cavity where collection is expected postoperatively.
- For drainage of empyema thoracis (pus in thoracic cavity).

30

Chapter

Wound Dressings and Bandages

Sanjay Marwah

DRESSING

It is a piece of material placed directly over the wound or ulcer to provide cover, comfort and support so as to encourage healing and to prevent infection from outside.

Parts of a Dressing

Contact Layer

It is made of sterile mesh gauze placed directly over the wound. It is non-absorbent and allows secretions to pass through its grid. It is made non-adherent by using mesh gauze soaked in petroleum jelly or using sofra-tulle so as to prevent pain and trauma during removal of the dressing.

Details of various agents used for wound dressing are given in chapter 5: Sinus, Fistula and Ulcer.

Intermediate Layer

It is made of fluffy cotton gauze sponges so that wound secretions are absorbed in this layer and do not reach up to the outermost layer. In case there is soaking of outer layer, it leads to bacterial migration into the wound from outside through capillary action apart from soiling of the clothes.

Outer Layer

It is the bandage that helps in supporting inner two layers and keeping them in persistent contact with the wound.

BANDAGES

- The technique of good and effective bandaging can be learnt only by repeated practice.
- A well-applied bandage gives immense relief and satisfaction to the patient.

- Cotton bandage is most commonly used as it is light weight and very economical. A variety of natural and synthetic materials can be used (crepe bandage, adhesive bandage, water proof bandage).
- The bandages are used for following purposes:
 - ☐ To stop bleeding by pressure.
 - ☐ To give rest and support to the affected part.
 - ☐ To prevent edema or swelling for which crepe bandage is often used.
 - ☐ To retain wound dressings in position.
 - ☐ To protect a surgical wound against infection.
 - ☐ To assist correction of a deformity.
- The width of the bandage is chosen according to the part to be bandaged (Box 30.1).
- Following **rules** are generally followed while applying the bandage:
 - ☐ Patient is placed in a comfortable position.
 - ☐ Part to be bandaged is put in natural position.
 - ☐ A tightly rolled bandage is used and only a short length (2-3") is unrolled at a time so as to maintain full control of the bandage.
 - ☐ The area distal to the bandaged part such as fingers or toes should be left exposed whenever possible so that it can be observed for any vascular impairment due to tight bandaging.
 - ☐ Bandage is applied in an upward direction and from within outward for a limb.

Box 30.1: Width of bandage based on part

Part to be bandaged	Width of the bandage
Finger/Toe	1"
Head	2"
Arm	3"
Leg	4"
Trunk	6"

- A few fixation turns are given in the beginning to firmly anchor the bandage.
- Each succeeding turn is given in a way that it overlaps two thirds of the preceding turn.
- Bandage is applied with even tension over the whole area.
- Pads are applied over the bony prominences.
- The bandage is finished with a complete turn and fixed with a strip of adhesive strapping, safety pin or with split and knotted end of the bandage.
- The mistakes to be avoided while applying a bandage are given in Box 30.2.

Box 30.2: Mistakes and their effects while applying a bandage

• Wet bandage	Shrinks on drying
• Very tight bandage	Interferes with circulation
• Loose bandage	Becomes displaced
• Reverse turns on a prominence	Pain and discomfort to the patient
• Too much cotton	Discomfort to the patient, expensive
• Too less cotton	Dressing gets soaked
• Incorrect securing of the knot	Pain and discomfort over injured area

- Following **turns** are used while applying roller bandages:
 - *Circular*: It is used for securing a bandage at the beginning and the end.
 - *Spiral*: When the bandage is carried spirally up the part with uniform thickness, e.g. finger, arm.
 - *Reverse spiral*: When the bandage is carried up the part with varying dimensions, e.g. forearm, leg (Fig. 30.1).
 - *Figure of eight*: It consists of overlapping turns each of which crosses at a mid-point and ascends or descends alternately. It is used over joints (Fig. 30.2).
 - *Spica*: It is modification of figure of eight turn when one loop is much larger than the other, e.g. one loop around upper thigh and second loop around lower trunk. It is so named because finished pattern resembles an ear of the wheat (Fig. 30.3).



Fig. 30.1: Reverse spiral bandage for the leg

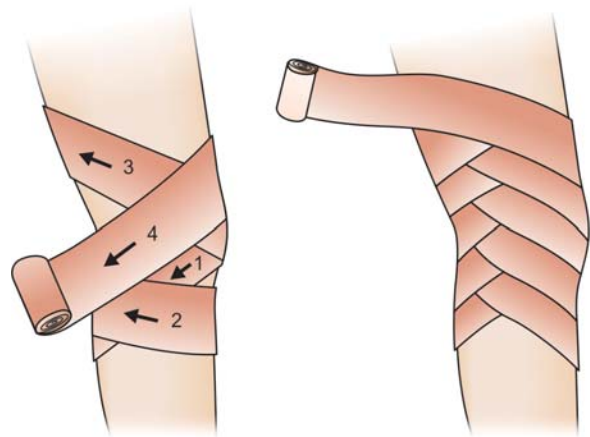


Fig. 30.2: Figure of eight bandage for the knee

- *Recurrent*: The bandage is laid over the area repeatedly by forward and backward turns. These turns are fixed with circular turns in the beginning and in the end, e.g. bandage to the head or to an amputation stump.

Bandaging for Head and Neck Region

Head Bandage

The two common ways to bandage the head are:

- (i) *Capeline bandage*: Take two bandages, (one 2" and second 2½" wide) and join their free ends in the middle of the forehead (Fig. 30.4). The surgeon stands behind the patient while holding two bandages in two hands

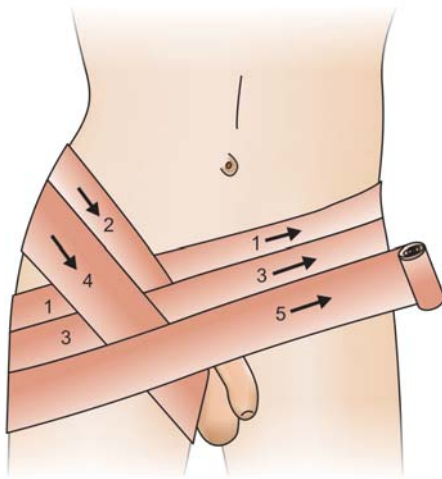


Fig. 30.3: Spica of the groin

that are rolled around the head from frontal to occipital region. Now the two bandages will have their independent courses, the wider one passing forwards or backwards over the head while the narrow bandage passes horizontally round the head fixing the wider

bandage. Finally with the narrow-bandage, two horizontal rounds are taken over the exhausted end of wider bandage and the end of narrow bandage is fixed with a safety pin.

(ii) *Recurrent bandage:* With a $2\frac{1}{2}$ " bandage, start a horizontal turn around the head starting just above the left ear. On reaching the starting point, make a reverse turn and guide the bandage over the center of the head to the right ear. Here reverse the bandage again and guide it back over center of the head to left ear. Pass the succeeding turns to and fro over the head first on one side and then on other side of the central bandage until head is covered. Finish with two horizontal turns round the head by tying the two free ends (Fig. 30.5).

Recurrent bandage is also used for covering amputation stump.

Eye Bandage

A $2\frac{1}{2}$ " wide bandage is placed against the forehead above the affected eye. It is passed around the head towards the sound eye above the ear and brought back low on occiput and forward to the starting point.

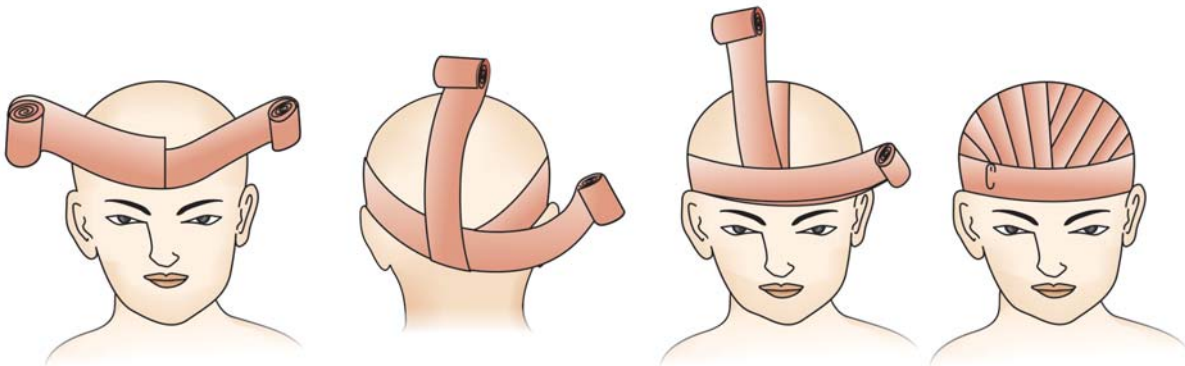


Fig. 30.4: Capeline bandage

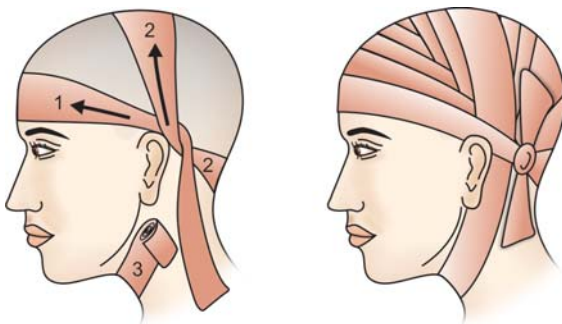


Fig. 30.5: Recurrent bandage



Fig. 30.6: Eye bandage

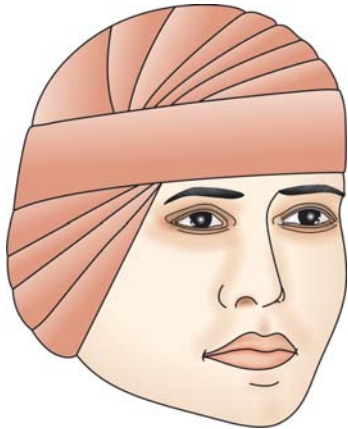


Fig. 30.7: Ear bandage

Hence, it takes second turn around the head and passes under the ear on the affected side and goes up covering the affected eye and then to center of the forehead. The bandage then continues obliquely around the head and multiple turns cover the affected eye. Finally a horizontal turn is made around the head and end of the bandage is secured over the forehead (Fig. 30.6).

Ear Bandage

It is same as eye bandage except that multiple oblique turns are made covering the affected ear. Insert cotton wool around the ear to prevent painful pressure points on the ear. This bandage is commonly used to cover mastoid region behind the ear following surgery on the mastoid bone (Fig. 30.7).

Bandage for Lower Jaw

In case of fracture of lower jaw, two types of bandages may be applied as first aid measure:

(i) *Barrel bandage*: A 3" wide bandage is taken and its center is placed under the chin. The two ends are brought over the head and tied with a single knot. The knot is then loosened to form a loop that is slipped and placed over the forehead in the front and occipital region on the back. The ends are then pulled tight and tied on the top of the head (Fig. 30.8).

(ii) *Four tailed bandage*: A 3" wide bandage is taken and its two ends are split longitudinally to make four tails. A

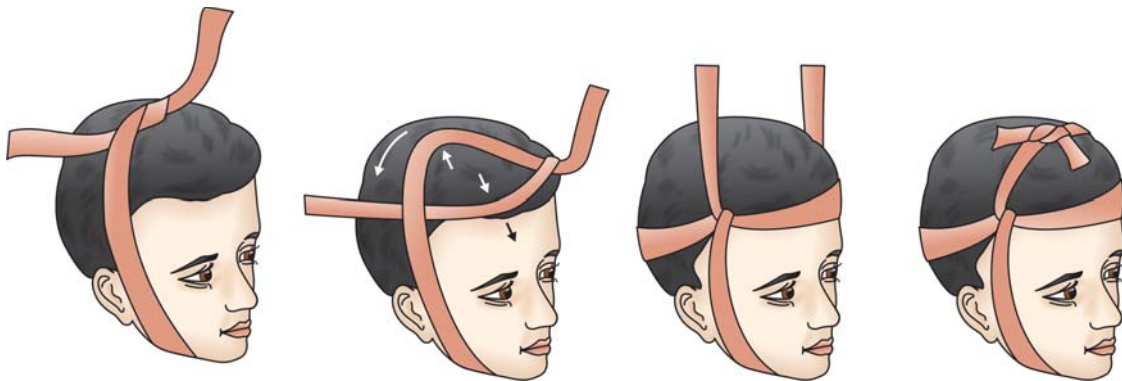


Fig. 30.8: Barrel bandage



Fig. 30.9: Four tailed bandage



Fig. 30.10: Neck bandage

hole is made in the middle of the bandage and hole bearing part of the bandage is applied to the chin. The two upper tails of the bandage are carried behind the neck and tied there. Similarly, the two lower tails are carried over the head and tied over the vertex. Finally the four tails are tied together behind the head (Fig. 30.9).

Neck Bandage

A 2½" wide bandage is rolled around the neck, carried across the chest, under the axilla, across the back, under the other axilla, over the chest, crossing the previous turn in the center of the chest. Thus, figure of eight turn is made around neck and upper chest. Multiple such turns are made till front of the neck is fully covered

(Fig. 30.10). A pad of cotton wool should be placed in each axilla, before starting the dressing.

This dressing is occasionally used to cover the thyroidectomy wound. Nowadays, thyroidectomy wound is sealed with a strip of adhesive tape put over a gauze piece since closed suction drain is being used for drainage of the wound.

Bandage for Fracture Clavicle

Figure of eight bandage is applied passing across the back, under the axilla and up in front of the shoulder and then crossing to the other side on the back of neck, opposite axilla and front of opposite shoulder. Cotton pads should be placed under the axillae before applying the bandage.

31

Chapter

Surgical Specimens

Nisha Marwah, Sanjay Marwah

To describe a surgical specimen:

- First of all examine the external surface to see any abnormality followed by examination of cut surface.
- Try to identify the parent organ if included in the specimen.
- Examine the pathological area of the specimen and try to identify the disease by various gross morphological features of the lesion.

PATHOLOGICAL SPECIMENS OF HEAD AND NECK

Important pathological specimens of head and neck include:

(The clinical details of these specimens are previously given in various chapters).

Tuberculous Lymphadenitis

- Commonly affects cervical lymph nodes.
- Majority of the patients are children and young adults.
- Neglected cases develop cold abscess that bursts to form discharging neck sinuses.

Gross: The lymph nodes are enlarged and matted together due to periadenitis. Cut-section reveals multiple yellow cheesy foci of caseous necrosis characteristic of tuberculosis (Fig. 31.1).

Microscopy:

Epithelioid cell granulomas
Langhans' giant cells
Foci of caseous necrosis

Differential diagnosis:

Lymphoma
Metastatic deposits
Reactive hyperplasia



Fig. 31.1: Cut section of matted lymph node mass showing central caseation

Diagnosis:

FNAC, lymph node biopsy.

Lymphoma

Hodgkin's lymphoma (Fig. 31.2):

- It is the malignant tumor of lymphoreticular system arising mostly in lymph nodes and rarely in extra nodal sites (liver, spleen etc.)
- It usually starts as painless enlargement of left supraclavicular lymph nodes.

Gross: Enlarged lymph node mass is pink gray with a homogeneous and fleshy cut surface.

Microscopy: Classical Reed-Sternberg cells are seen against a polymorphic background of lymphocytes, eosinophils, plasma cells, macrophages, etc.



Fig. 31.2: Hodgkin's lymphoma

Differential diagnosis:

- Tuberculous lymphadenitis
- Metastatic deposits
- Reactive hyperplasia.

Diagnosis

- Lymph node biopsy.

Multinodular Goiter

Gross: Features of multinodular goiter include asymmetric and extreme enlargement weighing up to 100-500 gm (normal weight 15-40 gm).

Cut section shows:

- Nodularity with poor encapsulation
- Fibrous scarring
- Hemorrhages
- Focal calcification
- Cystic degeneration.

Microscopy:

- Partial or incomplete encapsulation.
- Follicles of varying size.
- Areas of hemorrhages, fibrous scarring, calcification and cystic degeneration.
- Presence of hemosiderin—laden macrophages and cholesterol crystals.

Specimen 1: It shows two variably sized lobes connected with isthmus, an appearance characteristic of thyroid gland. Both the lobes and isthmus are

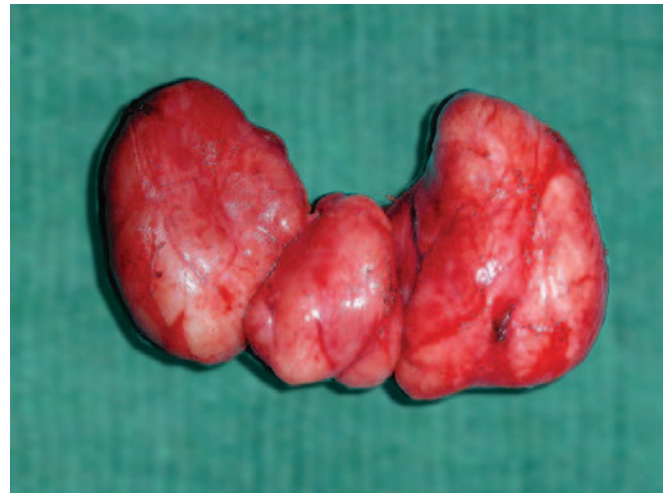


Fig. 31.3: Multinodular goiter operative specimen

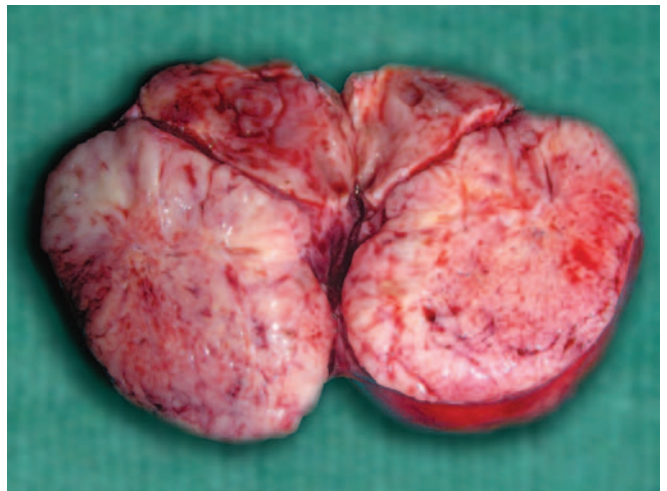


Fig. 31.4: Left lobectomy cut section

enlarged showing nodules of variable sizes suggestive of multinodular goiter (Fig. 31.3).

Specimen 2: Cut surface of the lobectomy specimen shows gray white fleshy appearance suggestive of adenomatous hyperplasia (Fig. 31.4).

Specimen 3: Cut surface of the lobectomy specimen shows brownish waxy appearance characteristic of colloid nodule (Fig. 31.5).

Squamous Cell Carcinoma of Mandible

Gross: Squamous cells carcinoma of oral cavity may have following gross types:

1. Ulcerative type: Indurated ulcer with everted edges.
2. Papillary/Verrucous type: Soft, friable and warty growth.

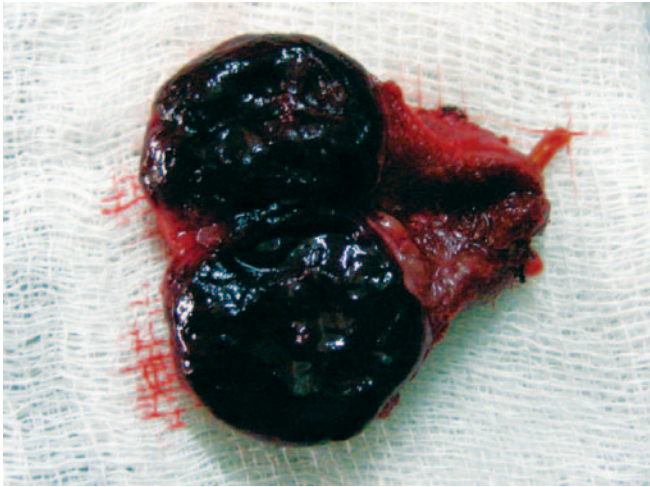


Fig. 31.5: Cut section of thyroid lobectomy specimen showing colloid filled nodule at lower pole



Fig. 31.6: Squamous cell carcinoma of mandible

3. Nodular type: Firm, submucosal nodule.
4. Scirrhus type: Infiltrative growth into deeper tissues.

Microscopy: Squamous cell carcinoma ranges from well-differentiated to poorly differentiated type. Malignant squamous epithelial nests and keratin pearls are seen.

Specimen: Hemimandibulectomy specimen shows a nodular and ulcerative growth involving ramus of the mandible (Fig. 31.6).



Fig. 31.7: Jaw tumor—osteosarcoma

Diagnosis: Wedge biopsy from margin of the lesion.

Osteosarcoma of Mandible

- Most common primary malignant tumor of the jaw.
- More commonly affects mandible than maxilla.
- Majority are intramedullary but may be parosteal.
- Prognosis for osteosarcoma of jaw is more favorable than for osteosarcoma of long bones.

Specimen: Gross examination of hemimandibulectomy specimen shows a large gray-white, firm, intramedullary tumor with near total involvement of mandible (Fig. 31.7).

Microscopy: Mostly have conventional appearance, i.e. sarcomatous stroma with osteoid formation.

Pleomorphic Adenoma

- Benign mixed salivary gland tumor.
- Most common in parotid gland usually involving superficial lobe.

Gross: The tumor is circumscribed, pseudo-encapsulated, multilobular and firm mass of variable size.

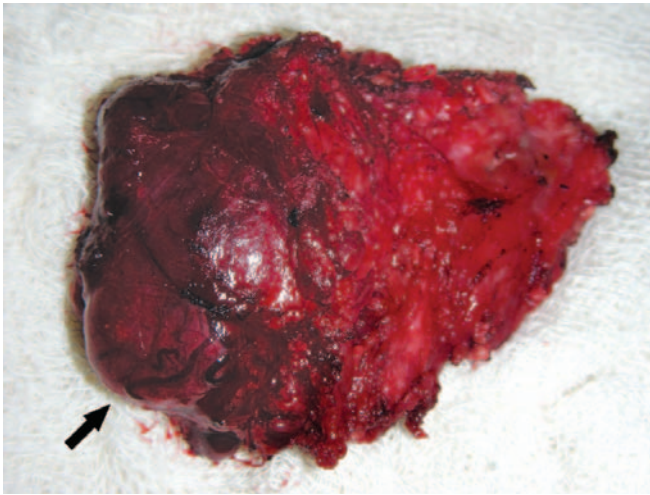


Fig. 31.8: Pleomorphic adenoma in superficial parotidectomy specimen—gross appearance

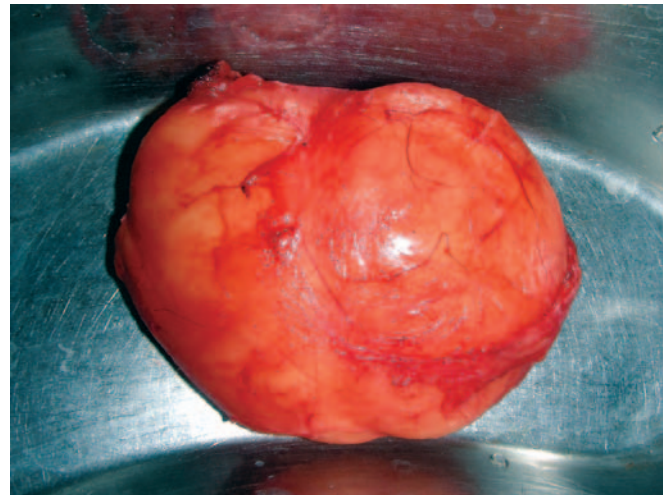


Fig. 31.10: Lipoma—gross appearance

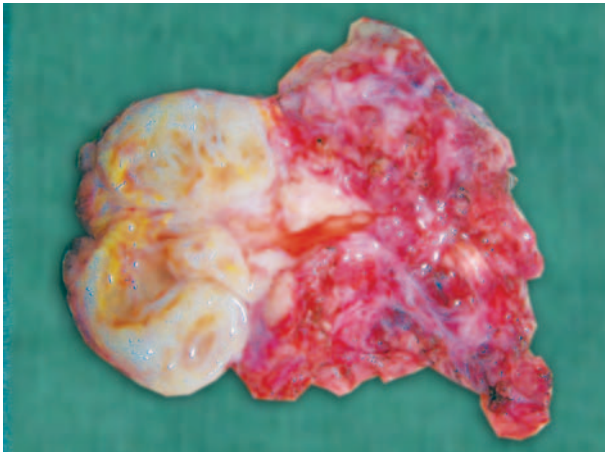


Fig. 31.9: Cut section of pleomorphic adenoma in superficial parotidectomy specimen

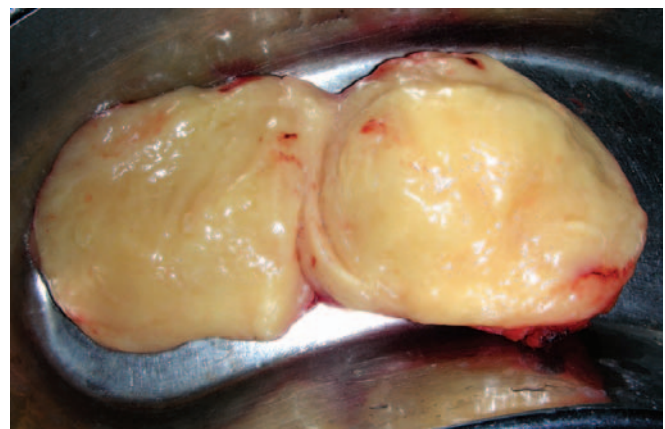


Fig. 31.11: Lipoma—cut section

Cut section shows gray-white, variegated appearance with translucent areas and soft to mucoid consistency.

Microscopy: Pleomorphic adenoma has two components:

1. Epithelial: Ductal and myoepithelial cells.
2. Mesenchymal: Muroid, myxoid and chondroid matrix.

Specimen: Superficial parotidectomy specimen shows pyramidal shaped parotid gland with a nodular mass involving lower portion of the gland (Fig. 31.8).

Cut surface shows a well-circumscribed tumor with pseudo-encapsulation and surrounding parotid gland. Cut section of tumor gives characteristic variegated and translucent appearance (Fig. 31.9).

Lipoma

- A benign tumor of adipose tissue that can occur anywhere (universal tumor).
- Commonest sites are nape of neck, abdominal wall and thigh.
- Large lipoma of thigh may rarely undergo malignant change.

Gross: The tumor is round to oval and encapsulated (Fig. 31.10). Cut surface is soft, lobulated, yellowish and greasy (Fig. 31.11).

Microscopy: The tumor is surrounded by a thin capsule. It is composed of lobules of mature adipose cells separated by thin fibrous septa.

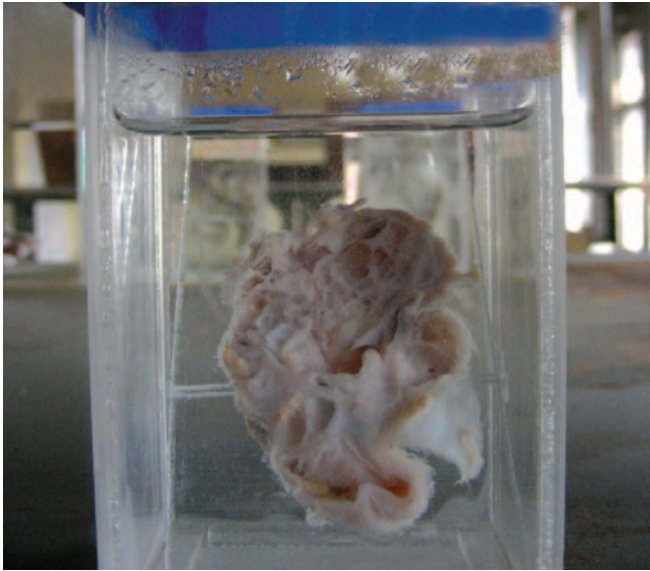


Fig. 31.12: Cystic hygroma—cut section

Cystic Hygroma

- Cystic hygroma is a multilocular swelling consisting of multiple cysts filled with clear lymph and lined by a single layer of epithelium.
- It is most commonly seen in neck region in children.
- The location of swelling is in lower third of neck in the posterior triangle.
- Most characteristic feature that distinguishes it from other similar swelling in neck is that it is *brilliantly transilluminant*.

Gross: Large, soft, spongy, multicystic mass containing cysts of variable sizes (Fig. 31.12).

Microscopy: Lesion consists of large dilated lymphatic spaces containing homogeneous pink lymph fluid.

Intervening stroma contains lymphoid infiltrate, sometimes lymphoid follicles.

Index

A

- ABG report 80
- ABO group 63
- Abrasion 46
- Abscess 16
 - pathophysiology 17
 - signs 17
 - symptoms 17
 - treatment 17
- Accessory nerve 194
- Acid-base balance and disorders 76
 - compensation in acid base disorders 77
 - anion gap 79
 - metabolic acidosis 78
 - metabolic alkalosis 79
 - respiratory acidosis 80
 - respiratory alkalosis 80
 - concept of pH 76
- Acidosis 80
- Acinic cell tumor 168
- Acquired bleeding disorders 65
- Acquired fistula 37
- Acquired response 9
- Acquired sinus 36
- Actinomycosis 29
 - clinical features 29
 - facio-cervical 29
 - liver 29
 - right iliac fossa 29
 - thoracic 29
 - diagnosis 30
 - treatment 30
- Actinomycosis 29,44,165
- Acute hemorrhage 8, 60
- Acute inflammation 12
- Acute laryngitis 176
- Acute lymphangitis 137
- Acute osteomyelitis 311
- Acute parotitis 164
- Acute retropharyngeal abscess 147
- Acute subdural hematoma 189
- Acute suppurative lymphadenitis 130
- Acute tonsillitis 145
- Adamantinoma 301
- Adenoid cystic carcinoma 168
- Adenoma 89
- Advanced laryngeal tumors 178
- Advantages of MRI 323
- Advantages of ultrasound 317
- Aerodigestive tract 321
- Aftercare of tracheostomy 182
- Aims of surgical repair 265
- Aims of tracheostomy 179
- Ainhum 215
- Airway 83
- Airway management 238
- Alkalosis 80
- Allis tissue forceps 345
- Alveolar abscess 310
- Ameloblastoma 301
- Analgesia 240
- Anaphylactic shock 68
- Anaplastic carcinoma 281
- Anatomical sinuses 36
- Anesthesia 236
 - advantages of local anesthesia 241
 - airway management 238
 - analgesia 240
 - central neuraxial blocks 243
 - chemical structure and classification 241
 - clearance 241
 - complications of local anesthesia 242
 - late complications 243
 - local complications 242
 - systemic complications 242
 - contraindications 242
 - equipment 238
 - endotracheal tubes (ETT) 238
 - face masks 238
 - flexible fiberoptic bronchoscope 238
 - laryngeal mask airway (LMA) 238
 - oral and nasal airways 238
 - rigid laryngoscope 238
 - extubation of trachea 240
 - general anesthesia 236
 - preanesthetic evaluation 237
 - premedication 237
 - preoperative fasting 237
 - indications of local anesthesia in dentistry 241
 - inhalational anesthetics 237
 - techniques of inhalation of anesthetics 237
 - intravenous induction agents 237
 - dissociative anesthesia 238
 - neurolept analgesia 238
 - local anesthesia 241
 - local anesthetic agents 242
 - local distribution 241
 - management of overdose reaction 243
 - mechanism of action 241
 - monitoring 240
 - neuromuscular blockers 240
 - pharmacological effects of local anesthetics 241
 - preparations of local anesthetics 241
 - routes of tracheal intubation 238
 - spinal anesthesia 243
 - stages of general anesthesia 237
 - technique 242
 - tracheal intubation 238
 - position of patient's head 238
- Aneurysm 117
- Aneurysm needle 348
- Angular stomatitis (angular cheilosis) 141
- Anion gap 79
- Ann Arbor staging 134
- Anterior pituitary 295
- Anthrax 29

diagnosis 29
 differential diagnosis 29
 treatment 29
 Antiboma 18
 Anticoagulant drugs 65
 Antithyroid drugs 276
 Aphthous stomatitis 140
 Aphthous ulcer 143
 Arterial (plexiform) angioma 116
 Arterial hemorrhage 59
 Arterial ulcer 42
 Artery forceps (hemostat) 346
 Atherosclerotic arterial thrombosis 200
 endovascular treatment 207
 percutaneous transluminal
 angioplasty (PTA) 207
 investigations 204
 management 204
 open surgery 206
 signs 201
 surgery for chronic lower limb
 ischemia 205
 symptoms 200
 Auditory nerve 194
 Autoclaving 57
 Autotransfusion 63
 Avulsion wounds 48
 Axonotmesis 192

B

Babcock's tissue forceps 345
 Bacteremia 20
 Bacterial infection 165
 Bandages 363
 bandaging for head and neck region
 364
 bandage for fracture clavicle 367
 bandage for lower jaw 366
 ear bandage 366
 eye bandage 365
 head bandage 364
 neck bandage 367
 Bard Parker knife handle 350
 Basal cell carcinoma (Rodent ulcer) 98
 Basal cell papilloma (Senile warts,
 seborrheic keratosis) 90
 Basophils 11
 Bell's palsy 196
 Benign neoplasms 148
 Benign tumors 89
 adenoma 89
 fibroma 89
 lipoma 90
 complications 91

diffuse lipoma 90
 encapsulated lipoma 90
 multiple lipomas 90
 neuroma 91
 elephantiasis neuromatosa 94
 false neuromas 91
 generalized neurofibromatosis (von
 Recklinghausen's disease) 92
 plexiform neurofibromatosis 93
 treatment of pigmented nevus 95
 true neuromas 91
 papilloma 89
 basal cell papilloma (Senile warts,
 seborrheic keratosis) 90
 squamous cell papilloma 89
 treatment 90
 Benign tumors 89,177, 277
 Bilateral neck nodes (N2C, N3) 157
 Biochemical investigations 293
 Biologic basis for dose fractionation
 234
 Black or hairy tongue 143
 Bleeding 59, 285
 external hemorrhage 59
 internal hemorrhage 59
 Bleeding disorders 65
 acquired bleeding disorders 65
 anticoagulant drugs 65
 hepatic failure 65
 hypothermia 65
 renal failure 65
 thrombocytopenia 65
 vitamin K deficiency 65
 congenital bleeding disorders 65
 investigations for bleeding
 disorders 65
 von Willebrand's disease 66
 Bleeding vessel 59
 arterial hemorrhage 59
 capillary hemorrhage 59
 venous hemorrhage 59
 Blood collection 62
 Blood grouping and cross matching 63
 Blood loss 59
 acute hemorrhage 60
 chronic hemorrhage 60
 mild hemorrhage 59
 moderate hemorrhage 60
 severe hemorrhage 60
 Blood storage 62
 Blood transfusion 62
 autotransfusion 63
 blood collection 62
 blood grouping and cross matching 63

ABO group 63
 Rh group 63
 blood storage 62
 complications of blood transfusion 63
 coagulation failure 64
 congestive heart failure 64
 immunosuppression 64
 infections 64
 problems of massive transfusion 64
 transfusion reactions 63
 fractions of blood 64
 transfusion of blood 63
 Blood vessels 187
 compartment of bleeding 187
 infratentorial hemorrhage 188
 supratentorial hemorrhage 188
 surgical anatomy of intracranial
 compartments 187
 management of head injury
 patient 189
 plane of bleeding 188
 Blood volume 62
 Boil (furuncle) 16
 Boiling 57
 Bone and joint 23
 Bone nibbler 354
 Bones 244
 cancellous bones 244
 tubular bones 244
 Brachytherapy 233
 Brain injury 186
 primary brain injury 186
 secondary brain injury 187
 Branchial cyst 120
 complications 121
 embryology 120
 pathology 120
 Branchial fistula 121
 differential diagnosis 122
 Breathing and ventilatory support 86
 Brodie's fistula director 354
 Buerger's disease 207
 investigations 208
 treatment 209
 chemical sympathectomy 209
 surgical sympathectomy 209
 Bupivacaine 242
 Burkitt's lymphoma 136
 Burns 326
 degrees of burns 326
 etiology 326
 management of burns 327
 early treatment 329
 emergency management 327
 first aid measures 327

local treatment 329
 nutrition 330
 surgical treatment 330
 Burns and skin grafting 326
 Bypass graft 206

C

Calcification 226
 Calculating acid-base status from ABG report 81
 Cancellous bones 244
 Cancrum oris 21
 treatment 21
 Capillary hemangioma 115
 Capillary hemorrhage 59
 Carbuncle 18
 clinical features 18
 treatment 18
 Carcinoma buccal mucosa (cheek) 151
 Carcinoma floor of mouth 150
 Carcinoma gingiva and lower alveolar ridge 151
 Carcinoma hard palate, upper alveolus and floor of maxillary antrum 155
 Carcinoma lip 152,155
 Carcinoma tongue 150
 Carcinoma tonsil 152, 155
 Carcinoma upper alveolar ridge, floor of maxillary antrum and hard palate 152
 Carcinomatous epulis 299
 Cardiogenic shock 68
 Care of the acutely injured 82
 Carotid body tumor (chemodectoma) (solid swelling) 118
 clinical features 118
 investigations 118
 treatment 118
 Cat's paw retractor 351
 Catarrhal inflammation 12
 Catheterization 362
 Cat-scratch disease 165
 Cavernous angioma 116
 Cellular events 9
 Cellulitis 19
 clinical features 19
 treatment 19
 Cellulitis in special sites 19
 neck 19
 clinical features 19
 treatment 20
 orbit 19
 Central neuraxial blocks 243

Cervical adenopathy and neck masses 320
 Cervical lymph nodes 127
 Burkitt's lymphoma 136
 causes of cervical lymphadenopathy 130
 acute suppurative lymphadenitis 130
 chronic nonspecific lymphadenitis 130
 glandular fever (infectious mononucleosis) 131
 secondary deposits in lymph nodes 131
 toxoplasmosis 131
 tuberculous lymphadenitis 131
 clinical examination of lymph nodes and lymphatic system 128
 general physical examination 128
 history 128
 local examination 128
 Hodgkin's lymphoma 133
 clinical features 133
 clinical staging (Ann Arbor staging) 134
 investigations 134
 treatment 135
 investigations 132
 operative steps of lymph node biopsy 132
 leukemia 136
 non-Hodgkin's lymphoma 135
 treatment 135
 surgical anatomy 127
 treatment 132
 role of chemotherapy 132
 types of neck dissection 132
 Cervical lymphadenopathy 130
 Cervical rib and thoracic outlet syndrome 211
 Chancre of the 144
 Characteristics of ideal suture material 338
 Cheate's forceps 344
 Chemical gangrene 215
 Chemical mediators of inflammation 9
 Chemical methods 57
 Chemical structure and classification 241
 Chemical sympathectomy 209
 Chemotherapy 157
 Chest and other parts 88
 Chronic dental sinus 310
 Chronic hemorrhage 60
 Chronic hyperplastic candidiasis 149

Chronic inflammation 13
 pathological features of chronic inflammation 13
 primary to chronic inflammation 13
 secondary to acute inflammation 13
 types of chronic inflammation 13
 chronic nonspecific inflammation 13
 chronic specific inflammation 13
 Chronic laryngitis 176
 Chronic lower limb ischemia 205
 Chronic nonspecific inflammation 13,130
 Chronic nonspecific ulcer 144
 Chronic osteomyelitis 312
 Chronic osteomyelitis associated with specific infection 313
 Chronic retropharyngeal abscess 147
 Chronic specific inflammation 13
 Chronic subdural hematoma 189
 Chronic tonsillitis 145
 Circulation and hemorrhage control 87
 Cirroid aneurysm 117
 Cleft lip and cleft palate 262
 aims of surgical repair 265
 anatomy of lip and palate 262
 associated anomalies 263
 classification 263
 complications of surgical repair 266
 embryology of lip and palate 262
 problems 264
 surgical repair of cleft lip 265
 steps of repair 265
 surgical repair of cleft palate 265
 time of surgical repair 265
 Clinical examination and differential diagnosis of a neck swelling 125
 Clinical features of various ulcers 42
 actinomycosis 44
 arterial ulcer 42
 diabetic ulcer 44
 Marjolin's ulcer 45
 neurogenic ulcer 43
 Rodent ulcer (basal cell carcinoma) 45
 squamous cell carcinoma 44
 syphilitic ulcer (gummatous ulcer) 44
 traumatic ulcer 42
 tropical ulcer (phagedenic ulcer) 44
 tubercular ulcer 44
 venous ulcer 42
 Clip applicator (Michel's) 349
 Clips extractor 350
 Cold abscess 26
 Compartment of bleeding 187
 Compartment syndrome 214

Compensation in acid-base disorders 77
 Compound fracture 244
 Concept of pH 76
 Condylar fracture 258
 Congenital bleeding disorders 65
 Congenital fissuring of the tongue 142
 Congenital fistula 37
 Congenital sinus 36
 Congenital syphilis 28
 Congestive heart failure 64
 Contact layer 363
 Contrast agents 323
 Control of bleeding 61
 Contusion 46
 Conventional radiography 315
 patient positioning 315
 Corrugated drain 356
 Course of events in diabetic foot 217
 Cracked lips 144
 Cranial nerves 192
 classification of nerve injuries 192
 Seddon classification 192
 examination of cranial nerves 193
 Cricoid hook 355
 Crush injury 213
 Crushed wounds 49
 Cryosurgery 232
 CT scan 318
 multislice or multidetector spiral CT 319
 principle 319
 reading a CT film 319
 advantages of CT scan 319
 spiral (helical) CT 319
 CT scan for head and neck lesions 320
 cervical adenopathy and neck masses 320
 nodal neck masses 321
 non-nodal neck masses 321
 head and neck tumors 320
 inflammatory lesions 320
 intracranial lesions 320
 masses arising from aerodigestive tract 321
 thyroid and parathyroid glands 321
 traumatic injuries 320
 vault and skull base lesions 320
 Curved scissors 350
 Cyst 107
 classification of cysts 107
 fluctuation test 108
 pulsations 109
 signs 107
 transillumination test 108
 complications in a cyst 110

Cystic hygroma 372
 Cystic swelling from lymphatics 119
 lymphangioma 119
 Cystic swellings from blood vessels 114
 aneurysm 117
 arterial (plexiform) angioma 116
 capillary hemangioma 115
 cirroid aneurysm 117
 hemangioma 114
 venous (cavernous) angioma 116
 Cysticercosis 124
 clinical features 124
 diagnosis 124
 treatment 124
 Cysts and neck swellings 107
 Cysts in mouth 142
 Cytopathological diagnosis 169
 Czyrny's retractor 352

D

Dangerous to life 51
 Deaver's retractor 352
 Deep vein thrombosis 226
 clinical features 226
 investigations 227
 treatment 227
 Degrees of burns 326
 Delivery systems for radiotherapy 233
 Dental cyst (radicular cyst, periodontal cyst) 300
 Dental ulcer 143
 Dentigerous cyst (follicular cyst) 300
 Depending upon nature of bleeding vessel 59
 Depending upon source of bleeding 59
 Depending upon speed of blood loss 60
 Depending upon time of hemorrhage 59
 Depending upon volume of blood loss 59
 Dermatitis 225
 Dermoid cyst 110
 types of dermoid 110
 implantation dermoid 112
 sequestration dermoid 110
 sublingual dermoid 112
 teratomatous dermoid 112
 tubulo-embryonic dermoid 113
 Detachable blades 350
 Developmental diseases 142
 Diabetic gangrene 217
 course of events in diabetic foot 217
 examination 218
 investigations 218

 pathophysiology 217
 treatment 218
 Diabetic ulcer 44
 Diathermy 230
 other energy sources used for tissue cutting and coagulation 232
 cryosurgery 232
 harmonic scalpel 233
 high frequency ultrasound waves 233
 lasers 232
 Differentiated thyroid cancer 279
 Differentiated thyroid carcinoma 278
 anaplastic carcinoma 281
 papillary carcinoma 280
 postoperative management 280
 treatment of differentiated thyroid cancer 279
 Diffuse toxic goiter 274
 Direct causes 213
 Directly observed treatment (DOT) for tuberculosis 26
 Disability 87
 Diseases of lymph nodes and lymphatics 127
 Diseases of oral cavity 140
 Diseases of venous system 220
 surgical anatomy of lower limb venous system 220
 surgical physiology 220
 Dislocation 244
 Dissecting forceps 345
 toothed forceps 345
 Dissection 228
 Dissector 354
 Donor site 331
 Doppler ultrasound 317
 Doyen's mouth gag 351
 Doyen's retractor 352
 Drains 356
 general principles for putting a drain 357
 types of drain 356
 Drawback of ultrasound 318
 Drawbacks of CT scan 320
 Drawbacks of MRI 323
 Dressing 363
 parts of a dressing 363
 contact layer 363
 intermediate layer 363
 outer layer 363
 Droplet infection 22
 Drugs causing gangrene 212
 ergot preparations 212
 intra-arterial drugs 212
 Dry heat 58

E

- Ear bandage 366
 - Early care 249
 - Early congenital syphilis 28
 - Early treatment 329
 - Ectopic thyroid 286
 - Edge 336
 - Elective tracheostomy 182
 - Electrolyte balance 72
 - Elephantiasis neuromatosa 94
 - Embolism 207
 - clinical features 207
 - Embryology 120
 - Embryology of lip and palate 262
 - Emergency management 327
 - Emergency tracheostomy 180
 - Encapsulated lipoma 90
 - Endocrine response 9
 - Endotracheal tubes (ETT) 238
 - Endovascular treatment 207
 - Environmental factors 97
 - Eosinophils 11
 - Epidemiology 166
 - Epiglottitis 175
 - causes 175
 - clinical features 175
 - treatment 175
 - Epulis 299
 - carcinomatous epulis 299
 - fibrous epulis 299
 - giant cell epulis (myeloid epulis) 299
 - granulomatous epulis (false epulis) 299
 - pregnancy epulis 299
 - Equipment 238
 - Ergot preparations 212
 - Erysipelas 20
 - treatment 20
 - Erythroplakia 149
 - Ethylene oxide (ETO) 58
 - Evaluation of patient 235
 - Examination of sinus/fistula 37
 - Exposure 87
 - External fistula 37
 - External hemorrhage 59
 - Extradural hematoma (EDH) 188
 - Extubation of trachea 240
 - Exudative inflammation 12
 - Eye 336
 - Eye bandage 365
 - Eye disease 287
-
- Face masks 238
 - Facial nerve 193
 - Facial nerve management 171
 - Facial nerve paralysis 195
 - causes of facial nerve paralysis 195
 - clinical features 195
 - Bell's palsy 196
 - investigations 196
 - surgical anatomy 195
 - treatment 196
 - Facial wounds 51
 - Facio-cervical 29
 - False neuromas 91
 - Fibroma 89
 - Fibrous epulis 299
 - First aid 246
 - First aid measures 327
 - Fistula 36
 - acquired fistula 37
 - congenital fistula 37
 - external fistula 37
 - internal fistula 37
 - Flap 331
 - Flexible fiberoptic bronchoscope 238
 - Fluctuation test 108
 - Fluid therapy 74
 - Follicular carcinoma 279
 - Foot deformity 226
 - Forceps used for hemostasis 346
 - artery forceps (hemostat) 346
 - large artery forceps 347
 - medium artery forceps 346
 - small or mosquito forceps 346
 - Kocher's forceps 347
 - Pott's bulldog clamp 348
 - Well's arterial clamp 348
 - Foreign body giant cells 12
 - Formaldehyde 58
 - Fournier's gangrene 216
 - Fractions of blood 64
 - Fracture 244
 - Fracture clavicle 367
 - Fracture mandible 251
 - Fracture maxilla 252
 - Fracture of nasal bones 252, 254
 - Fracture of the mandible 255
 - condylar fracture 258
 - fracture of non-tooth bearing segment 257
 - gunning splint 257
 - fracture of tooth bearing segment 255
 - closed reduction with indirect fixation 255
 - open reduction with internal fixation 257
 - patterns of mandible fracture 255
 - treatment of mandible fracture 255
 - Fracture of maxilla 258
 - complications of maxillofacial fractures 260
 - orbital blow out fracture 260
 - patterns of fracture maxilla 258
 - treatment of fracture maxilla 259
 - Fracture of the zygomatic complex 253
 - Fracture of tooth bearing segment 255
 - Fracture treatment 248
 - Fracture zygomatic arch 252
 - Fractures 244
 - clinical examination 250
 - clinical features and diagnosis 246
 - examination 246
 - history 246
 - radiological examination 246
 - complications of fractures 249
 - compound fracture 244
 - early care 249
 - fractures of head and neck region 249
 - healing of a fracture 245
 - management 246
 - definitive management 247
 - first aid 246
 - general management of patient 247
 - immobilization 247
 - local management of fracture 247
 - reduction 247
 - rehabilitation 247
 - treatment of open fractures 248
 - treatment of uncomplicated closed fractures 247
 - maxillofacial fractures—classification 249
 - newer methods of fracture treatment 248
 - radiological investigations 251
 - simple fracture 244
 - treatment 252
 - fracture of nasal bones 254
 - fracture of zygomatic complex 253
 - general measures 252
 - Fractures and maxillofacial fractures 244
 - dislocation 244
 - fracture 244
 - sprain 244
 - subluxation 244
 - Fractures of head and neck region 249
 - Frey's syndrome 171
 - Frostbite 214
 - Full thickness graft 332

F

G

Gamma irradiation 58
 Gangrene 198
 causes of gangrene 198
 individual causes of gangrene 200
 spread of gangrene 199
 treatment of gangrene 200
 Gangrene and diseases of arterial system 198
 Gangrenous stomatitis (cancrum oris) 142
 General measures 69
 Geographic tongue (glossitis migrans) 143
 Giant cell epulis (myeloid epulis) 299
 Giant cells 11
 Gigli saw 353
 Gland tumors 169
 Glandular carcinoma 106
 Glandular fever (infectious mononucleosis) 131
 Globulomaxillary cyst 302
 Glossopharyngeal nerve 194
 Goiter 270
 complications 272
 diffuse hyperplastic goiter 271
 investigations 272
 multinodular goiter (MNG) 271
 retrosternal goiter 273
 stages in goiter formation 271
 symptoms and signs 271
 treatment 273
 Goiter formation 271
 Gonorrhea 28
 diagnosis 28
 treatment 28
 complications 28
 Graft loss 333
 Grafting 331
 Granny knot 339
 Granulomatous epulis (false epulis) 299
 Granulomatous sialadenitis 165
 Granulomatous thyroiditis 284
 Graves' disease 275
 Grievous injury 51
 Gunning splint 257

H

Harmonic scalpel 233
 Hashimoto's thyroiditis 284
 Head and neck lesions 315
 Head and neck region 95
 Head and neck tumors 320

Head and scalp/maxillofacial examination 88
 Head bandage 364
 Head injury 184
 classification 184
 mechanisms 184
 primary injury 184
 secondary injury 184
 Head injury and cranial nerves injury 184
 Head injury patient 189
 Healing 327
 Healing and organization 13
 Healing of a fracture 245
 Hemangioma 114
 Hematological investigations 6
 Hematoma 46
 Hemophilia 65
 Hemorrhage 59, 61, 182, 226
 control of bleeding 61
 operative methods 62
 position 62
 pressure and packing 61
 primary hemorrhage 59
 reactionary hemorrhage 59
 restoration of blood volume 62
 secondary hemorrhage 59
 Hemorrhage, blood transfusion and bleeding disorders 59
 Hemostasis 229
 Hepatic failure 65
 Herpes stomatitis 141
 High frequency ultrasound waves 233
 Histiocytosis X 308
 Hodgkin's lymphoma 133
 Hudson's brace and burr 353
 Humby skin grafting knife 355
 Hypercalcemia 294
 clinical features 294
 etiology 294
 pathophysiology 294
 treatment 294
 Hyperkalemia 72
 Hyponatremia 72
 Hyperparathyroidism 292
 clinical features 292
 diagnosis 293
 biochemical investigations 293
 radiological investigations 293
 differential diagnosis 293
 primary hyperparathyroidism 292
 secondary hyperparathyroidism 292
 tertiary hyperparathyroidism 292
 Hyperpituitarism 295
 Hyperplastic goiter 271

Hypervolemia 71
 Hypocalcemia 285
 Hypoglossal nerve 194
 Hypokalemia 73
 Hyponatremia 72
 Hypoparathyroidism 291
 clinical features 291
 diagnosis 292
 treatment 292
 Hypopituitarism 296
 Hypothalamic—pituitary thyroid axis 268
 Hypothermia 65
 Hypothyroidism 269, 285
 causes 269
 investigations 270
 myxoedema 270
 signs (starting from head to toe) 270
 symptoms 270
 treatment 270
 Hypovolemia 71
 Hypovolemic shock 67

I

Imaging techniques for head and neck lesions 315
 Immobilization 247
 Immune response 9
 Immunosuppression 64
 Implantation dermoid 112
 Incised wounds 48
 Indirect causes 214
 Indirect fixation 255
 Inducible nitric oxide synthase (nos-2) and cyclooxygenase-2 11
 Infant feeding tube 358
 Infections 15
 definition 15
 pathophysiology 15
 Infectious mononucleosis (glandular fever) 147
 Infective gangrene 215
 clinical features 215
 pathogenesis 215
 treatment 216
 Inflammation 8
 acute inflammation 8
 cellular events 9
 vascular events 8
 chronic inflammation 8
 signs of inflammation 8
 types of inflammation 8
 Inflammatory cells 11
 basophils 11

- eosinophils 11
 - giant cells 11
 - foreign body giant cells 12
 - Langhans' giant cells 12
 - Reed-Sternberg cells 12
 - lymphocytes 11
 - macrophages 11
 - neutrophils 11
 - plasma cells 11
 - Inflammatory disorders 163
 - acute parotitis 164
 - bacterial infection 165
 - obstructive causes 165
 - viral infection 164
 - granulomatous sialadenitis 165
 - actinomycosis 165
 - cat-scratch disease 165
 - sarcoidosis 165
 - tuberculosis 165
 - pseudoparotomegaly 166
 - sialadenosis 166
 - submandibular sialadenitis 163
 - clinical features 163
 - complications 164
 - diagnosis 164
 - differential diagnosis 164
 - treatment 164
 - Inflammatory lesions of tongue 143
 - Inflammatory swellings 310
 - chronic dental sinus 310
 - osteomyelitis of jaws 311
 - acute osteomyelitis 311
 - chronic osteomyelitis 312
 - necrosis of the jaw 313
 - pericoronitis 310
 - complications of alveolar abscess 310
 - Infratentorial hemorrhage 188
 - Ingestion 22
 - Inhalational anesthetics 237
 - Injury to blood vessels 187
 - Instruments used for cleaning and draping 344
 - Cheatle's forceps 344
 - Mayo's towel clip 344
 - Moynihan's tetra-towel clip 345
 - Ramsey's sponge holding forceps 344
 - Instruments used for making incision 350
 - Bard Parker knife handle 350
 - Detachable blades 350
 - Interferon-g 11
 - Interleukin-1 10
 - Intermediate layer 363
 - Internal fistula 37
 - Internal fixation 257
 - Internal hemorrhage 59
 - Interventional radiology 324
 - non-vascular 324
 - vascular 324
 - Intestinal tuberculosis 23
 - Intra-arterial drugs 212
 - Intracerebral and intraventricular hemorrhage 189
 - Intracranial compartments 187
 - Intracranial lesions 320
 - Intraoperative analgesia 240
 - Intraoperative complications 182
 - Intravenous induction agents 237
 - Introduction of surgery 1
 - dealing with a surgical patient 3
 - examination 5
 - history of surgery 1
 - history taking 4
 - investigations 6
 - management of unfit patient 7
 - risk assessment of the surgery 7
 - Investigations 123
 - Ionizing radiations 234
 - Ipsilateral neck nodes 157
 - Isotope scan 269
-
- ### J
-
- Jaws 314
 - examination of maxilla 314
 - examination of mandible 314
 - Joffroy's sign 288
 - Joll's thyroid retractor 351
-
- ### K
-
- Keratoacanthoma (molluscum sebaceum) 95
 - Kocher's forceps 347
 - Kocher's thyroid retractor 351
-
- ### L
-
- Lacerated wounds 48
 - Laceration of tongue 143
 - Lane's tissue forceps 346
 - Langenbeck's retractor 352
 - Langhans' giant cells 12
 - Large artery forceps 347
 - Laryngeal mask airway 238
 - Laryngectomy 178
 - Laryngitis 176
 - acute laryngitis 176
 - chronic laryngitis 176
 - laryngocele 176
 - vocal cord polyp 176
 - Laryngocele 123
 - Larynx 175, 177
 - physiology 175
 - surgical anatomy 175
 - Lasers 232
 - Late congenital syphilis 28
 - Latent syphilis 27
 - Le Fort I fracture (horizontal fracture) 258
 - Le Fort II fracture (pyramidal fracture) 259
 - Le Fort III fracture (craniofacial dysjunction) 259
 - Leprosy (Hansen's disease) 32
 - classification 32
 - clinical features 32
 - diagnosis 33
 - treatment 33
 - Leukoplakia 148
 - Lichen planus 143
 - Lidocaine 242
 - Life history of an ulcer 39
 - Lingual thyroid 142
 - Lip and palate 262
 - Lipodermatosclerosis 225
 - Lipoma 90
 - Lips 144
 - chancere of lip 144
 - cracked lips 144
 - macrocheilia 144
 - pigmented lips 144
 - Liver 29
 - Local anesthesia 242
 - Local anesthesia in dentistry 241
 - Lower jaw 366
 - Lower limb venous system 220
 - Lupus vulgaris 23
 - Lymph node biopsy 132
 - Lymph nodes 131
 - Lymph nodes and lymphatic system 128
 - Lymph nodes and lymphatics 127
 - Lymphangioma 119
 - Lymphangitis 20
 - Lymphatic system 136
 - acute lymphangitis 137
 - anatomy of lymphatic system 136
 - lymphedema 137
 - clinical features of lymphedema 138
 - investigations 138
 - primary lymphedema 137
 - secondary lymphedema 137
 - physiology of lymphatic system 137
 - Lymphatic system 137
 - Lymphedema 137, 138
 - Lymphocytes 11
 - Lymphoma 106

M

- Macrocheilia 144
- Macroglossia 143
- Macrophages 11
- Magnetic resonance imaging 322
 - contrast agents 323
 - principle 322
 - radiofrequency sequences 322
 - T1 and T2 weighted images 322
- Malecot's catheter 362
- Malignant lymphoma 282
- Malignant melanoma 103
- Malignant neoplasms 148
- Malignant non-osseous tumors 307
- Malignant osseous tumors 306
- Malignant tumors 95, 177, 277
 - basal cell carcinoma (Rodent ulcer) 98
 - etiology 96
 - glandular carcinoma 106
 - lymphoma 106
 - malignant melanoma 103
 - sarcoma 97
 - spread of malignant tumors 97
 - squamous cell carcinoma (epithelioma, epidermoid carcinoma) 100
 - clinical features 100
 - spread 101
 - treatment 101
 - types 100
 - staging of malignant tumors 97
 - tumor grading 97
 - types of malignant tumors 97
- Malignant tumors of maxilla 309
- Malignant ulcer 144
- Malleable probe 354
- Mandible 314
- Mandible fracture 255
- Marjolin's ulcer 45, 226
- Massive transfusion 64
- Maxilla 314
- Maxillofacial fractures 249, 260
- Mayo's towel clip 344
- Median cyst 302
- Median rhomboid glossitis 142
- Medicolegal aspects of wounds 51
- Medium artery forceps 346
- Medullary carcinoma 281
- Meleney's gangrene (pyoderma gangrenosum) 216
- Meningocele 124
 - clinical features 124
 - complications 124
 - treatment 125
- Mesh graft 333
- Mesothelial odontomes 302
- Metabolic acidosis 78
- Metabolic alkalosis 79
- Metallic tube 356
- Metastatic deposits 308
- Metastatic nodes—secondary to
 - unknown primary tumor 132
- Methods for determining blood loss 60
- Mild hemorrhage 59
- Miliary tuberculosis 23
- Minor salivary gland tumors 169
- Miscellaneous instruments 353
 - Bone nibbler 354
 - Brodie's fistula director 354
 - Gigli saw 353
 - Hudson's brace and burr 353
 - Humby skin grafting knife 355
 - Kocher's thyroid dissector 355
 - malleable probe 354
 - Myer's vein stripper 355
 - periosteal elevator 353
 - probe with dissector 354
 - trocár and cannula 353
 - Volkman's scoop (curette) 355
- Moebius sign 288
- Monilial stomatitis (thrush) 141
- Morris retractor 352
- Mouth 140
 - cysts in the mouth 142
 - solitary oral ulcer 142
 - stomatitis 140
 - angular stomatitis (angular cheilosis) 141
 - aphthous stomatitis 140
 - gangrenous stomatitis (cancrum oris) 142
 - herpes stomatitis 141
 - monilial stomatitis (thrush) 141
 - ulcerative stomatitis (Vincent's angina) 141
 - submucous fibrosis 142
 - syphilis 142
- Moynihan's tetra-towel clip 345
- MRI for head and neck lesions 323
- MRI vs CT scan 323
 - advantages of MRI 323
 - drawbacks of MRI 323
- MRI for head and neck lesions 323
- Mucoepidermoid tumor 168
- Mucous retention cyst 162
- Multinodular goiter (MNG) 271, 369
- Multiple drug resistance (MDR)
 - tuberculosis 25
- Multiple lipomas 90
- Multiple myeloma 308

- Multislice or multidetector spiral CT 319
- Myelocele 125
- Myer's vein stripper 355
- Myxoedema 270

N

- N0 nodes 157
- Nasolabial cyst 302
- Nasopalatine cyst 302
- Neck and cervical spine examination 88
- Neck bandage 367
- Neck dissection 132
- Neck nodes 171
- Neck swelling 125
- Necrosis of the jaw 313
- Needle 336, 348
- Needle holder 348
- Neoplasms 277
- Neoplastic lesions of oral cavity 148
 - benign neoplasms 148
 - malignant neoplasms 148
 - incidence 148
 - prognosis 148
 - premalignant lesions in oral cavity 148
 - chronic hyperplastic candidiasis 149
 - erythroplakia 149
 - leukoplakia 148
 - oral submucous fibrosis 149
 - syphilitic glossitis 149
- Nerve hook/retractor 351
- Nerve injuries 192
- Neurofibroma 91
- Neurofibromatosis (von Recklinghausen's disease) 92
- Neurogenic shock 68
- Neurogenic ulcer 43
- Neurolept analgesia 238
- Neuromuscular blockers 240
- Neuropraxia 192
- Neurotmesis 192
- Neutrophils 11
- Nodal neck masses 321
- Non-Hodgkin's lymphoma 135
- Non-nodal neck masses 321
- Non-osseous jaw tumors 307
- Non-toothed or plain forceps 345
- Nutrition 330

O

- Oculomotor (III), trochlear (IV) and abducent (VI) nerves 193
- Odontogenic and non-odontogenic cysts 125

- Odontogenic keratocyst 301
 Odontomes 300
 adamantinoma (ameloblastoma) 301
 dental cyst (radicular cyst, periodontal cyst) 300
 dentigerous cyst (follicular cyst) 300
 mesothelial odontomes 302
 globulomaxillary cyst 302
 median cyst 302
 nasolabial cyst 302
 nasopalatine cyst 302
 solitary bone cyst (hemorrhagic or aneurysmal bone cyst) 302
 odontogenic keratocyst 301
 Olfactory nerve 193
 Open fractures 248
 Operation theater discipline 54
 Operative surgery 228
 dissection 228
 hemostasis 229
 skin incision 228
 wound closure 229
 Operative surgery, diathermy, radiotherapy and anesthesia 228
 Optic nerve 193
 Oral and nasal airways 238
 Oral cancers 150, 152, 153
 carcinoma buccal mucosa (cheek) 151
 carcinoma floor of mouth 150
 carcinoma gingiva and lower alveolar ridge 151
 carcinoma lip 152
 carcinoma tongue 150
 carcinoma tonsil 152
 carcinoma upper alveolar ridge, floor of maxillary antrum and hard palate 152
 investigations for oral cancers 152
 ipsilateral neck nodes 157
 bilateral neck nodes (N2C, N3) 157
 prognosis 157
 staging of oral cancers 153
 treatment of oral cancers 153
 carcinoma hard palate, upper alveolus and floor of maxillary antrum 155
 carcinoma lip 155
 carcinoma tonsil 155
 chemotherapy 157
 radiotherapy 156
 surgery 153
 treatment of neck nodes 157
 treatment of primary tumor 153
 Oral cavity 140, 148, 158
 examination 158
 history 158
 inspection 158
 palpation 59
 Oral submucous fibrosis 149
 Orbit 19
 Orbital blow out fracture 260
 Osseous jaw tumors 303
 Osteomyelitis of jaws 311
 Osteosarcoma of mandible 370
 Overdose reaction 243
-
- P**
-
- Palatal perforations 144
 Palatal swellings 144
 Palate 144
 palatal perforations 144
 palatal swellings 144
 Papillary carcinoma 278,280
 Parapharyngeal abscess 146
 Parathyroid and pituitary gland 291
 Parathyroid gland 291
 physiology 291
 surgical anatomy 291
 Parotid gland tumors 167
 Parotidectomy 171
 Paterson-Kelly syndrome 149
 Pathological specimens of head and neck 368
 cystic hygroma 372
 lipoma 371
 lymphoma 368
 multinodular goiter 369
 osteosarcoma of mandible 370
 pleomorphic adenoma 370
 squamous cell carcinoma of mandible 369
 tuberculous lymphadenitis 368
 Patterns of fracture 245
 Pedicle skin flap 333
 Percutaneous transluminal angioplasty (PTA) 207
 Pericoronitis 310
 Periosteal elevator 353
 Periostitis 226
 Peripheral neuropathy 217
 Peripheral vascular disease 217
 Peritonsillar abscess (quinsy) 146
 PET scan 324
 Pharmacological effects of local anesthetics 241
 Pharyngeal pouch 122
 clinical features 122
 investigations 123
 surgical anatomy 122
 treatment 123
 Physical gangrene 214
 ainhum 215
 frostbite 214
 treatment 215
 trench foot 215
 Pigmented lips 144
 Pigmented nevus 95
 Pituitary gland 295
 hyperpituitarism 295
 hyperfunction of anterior pituitary 295
 hyperfunction of posterior pituitary and hypothalamus 296
 hypopituitarism 296
 hypofunction of anterior pituitary 296
 hypofunction of posterior pituitary and hypothalamus 296
 pituitary tumors 296
 clinical features 297
 investigations 297
 treatment 297
 Pituitary hormones 295
 hormones of anterior pituitary 295
 hormones of posterior pituitary 295
 Pituitary tumors 296
 Plane of bleeding 188
 Plasma cells 11
 Plastic and rubber instruments 357
 infant feeding tube 358
 Malecot's catheter 362
 Ryle's tube 357
 uses of Ryle's tube 358
 urethral catheters 359
 complications of catheterization 362
 technique of catheterization 361
 types 359
 venesection cannula 358
 indications of venous cut down 359
 Plastic tubes 356
 Pleomorphic adenoma 167,370
 Plexiform neurofibromatosis 93
 Plummer-Vinson syndrome 149
 Posterior pituitary and hypothalamus 296
 Postoperative analgesia 240
 Postpertussis ulcer 144
 Potato nose (rhinophyma) 95
 Pott's bulldog clamp 348
 Pregnancy epulis 299

Prehospital management and first aid of
 trauma patients 82
 transport 83
 treatment 82
 triage 82
 Pressure sores (bed sores /trophic
 ulcers) 213
 Principles of operative surgery,
 diathermy, radiotherapy and
 anesthesia 228
 Profundaplasty 207
 Pseudomembranous inflammation 12
 Pseudoparotomegaly 166
 Pulmonary tuberculosis 22
 Punctured wounds 48
 Putting a drain 357
 Pyemia 21

R

Radiation dosage (dosimetry) 234
 Radical neck dissection 132, 133
 Radiofrequency sequences 322
 Radioiodine 277
 Radionuclide studies 324
 Radiotherapy 156, 171, 233
 biologic basis for dose
 fractionation 234
 clinical applications of
 radiotherapy 235
 pretreatment evaluation of
 patient 235
 treatment goals 235
 delivery systems for radiotherapy 233
 brachytherapy 233
 targeted therapy 234
 teletherapy 233
 radiation dosage (dosimetry) 234
 technical considerations 236
 toxicity 236
 types of ionizing radiations 234
 Rampley's sponge holding forceps 344
 Ranula 163
 Rare causes 274
 Raynaud's disease 210
 complications 210
 pathophysiology 210
 treatment 210
 Raynaud's syndrome 211
 treatment 211
 Reed-Sternberg cells 12
 Reef knot 338
 Reidel's thyroiditis 284
 Retractors 351
 Cat's paw retractor 351
 Czyrny's retractor 352

Deaver's retractor 352
 Doyen's mouth gag 351
 Doyen's retractor 352
 Joll's thyroid retractor 351
 Kocher's thyroid retractor 351
 Langenbeck's retractor 352
 Morris retractor 352
 nerve hook/retractor 351
 self-retaining abdominal wound
 retractor 352
 skin hook 351
 Retropharyngeal abscess 147
 Retrosternal goiter 273
 Rh group 63
 Right iliac fossa 29
 Rigid laryngoscope 238
 Rodent ulcer (basal cell carcinoma) 45
 Rt in pleomorphic adenoma 171
 Ryle's tube 357, 358

S

Salivary gland tumors 166
 acinic cell tumor 168
 adenoid cystic carcinoma 168
 complications of parotidectomy 171
 epidemiology 166
 facial nerve management 171
 Frey's syndrome 171
 histological classification 166
 history 172
 investigations for salivary gland
 tumors 169
 cytopathological diagnosis 169
 radiological evaluation 169
 management of neck nodes 171
 minor salivary gland tumors 169
 mucoepidermoid tumor 168
 parotid gland tumors 167
 pleomorphic adenoma 167
 role of radiotherapy 171
 Rt in pleomorphic adenoma:
 indications 171
 Sjögren's syndrome 172
 submandibular gland tumors 169
 superficial parotidectomy 170
 treatment of salivary gland
 tumors 169
 Warthin's tumor 168
 Salivary glands 161, 162, 172
 mucous retention cyst 162
 parotid gland 161
 ranula 163
 sublingual gland 162
 submandibular gland 162
 surgical anatomy 161

Saphenous vein 224
 Sarcoidosis 165
 Sarcoma 97
 Scalp laceration 184
 Scarring and keloid formation 286
 Scissors 350
 curved scissors 350
 straight scissors 350
 Sebaceous cyst 113
 clinical features 113
 complications 113
 Secondary brain injury 187
 Seddon classification 192
 Selective neck dissection 133
 Self-retaining abdominal wound
 retractor 352
 Septicemia 21
 prevention 21
 treatment 21
 Sequestration dermoid 110
 Shape 336
 Shock 67
 definition 67
 pathophysiology 67
 treatment of shock 69
 types of shock 67
 anaphylactic shock 68
 cardiogenic shock 68
 hypovolemic shock 67
 neurogenic shock 68
 septic shock 69
 Shock, water-electrolyte and acid-base
 balance 67
 Short saphenous vein 224
 Sialadenitis 163, 166
 Sideropenic dysphagia 149
 Signs (starting from head to toe) 270
 Sinus 36
 acquired sinus 36
 anatomical sinuses 36
 congenital sinus 36
 Sinus, ulcer and fistula 36
 Sinus/fistula 37
 Sjögren's syndrome 172
 Skin grafting 331
 donor site 331
 causes of graft loss 333
 methods of grafting 331
 flap 331
 full thickness graft 332
 grafting 331
 pedicle skin flap 333
 mesh graft 333
 stamp graft 333
 recipient site 331
 Skin hook 351

- Skin incision 228
 Skull fractures 185
 Small or mosquito forceps 346
 Solitary bone cyst (hemorrhagic or aneurysmal bone cyst) 302
 Solitary oral ulcer 142
 Solitary thyroid nodule 282
 Special types of acute inflammation 12
 catarrhal inflammation 12
 exudative inflammation 12
 outcome of acute inflammation 12
 chronic inflammation 13
 healing and organization 13
 resolution 12
 suppuration 13
 pseudomembranous inflammation 12
 treatment of acute inflammation 13
 ulceration 12
 Specific infections 22
 Spect 325
 Spiral (helical) CT 319
 Sprain 244
 Squamous cell carcinoma (epithelioma, epidermoid carcinoma) 100
 Squamous cell carcinoma of mandible 369
 Squamous cell papilloma 89
 Stamp graft 333
 Stelwag's sign 287
 Sterilization 56
 autoclaving 57
 boiling 57
 chemical methods 57
 dry heat 58
 ethylene oxide (ETO) 58
 formaldehyde 58
 gamma irradiation 58
 Sternomastoid tumor (solid swelling) 118
 Stomatitis 140
 Straight scissors 350
 Stridor 175
 Subdural hematoma (SDH) 189
 Subhyoid bursal cyst 123
 Sublingual dermoid 112
 Sublingual gland 162
 Submandibular gland 162
 Submucous fibrosis 142
 Superficial parotidectomy 170
 Suppuration 13
 Supratentorial hemorrhage 188
 Surface epithelium 49
 Surgeon's knot 339
 Surgery 1, 153
 Surgical asepsis and antiseptic measures 54
 Surgical instruments 344
 Surgical knots 338
 Surgical needles 336
 classification 336
 parts of a needle 336
 Surgical repair 266
 Surgical specimens 368
 Surgical suturing 336
 Surgical sympathectomy 209
 Survey 87
 Suture materials 337
 characteristics of ideal suture material 338
 classification 337
 principles for selecting sutures 337
 size of sutures 337
 Sutures 337
 Suturing instruments 348
 aneurysm needle 348
 clip applicator (Michel's) 349
 clips extractor 350
 needle holder 348
 surgical needles 348
 Suturing techniques 338
 types of surgical knots 338
 Granny knot 339
 Reef knot 338
 surgeon's knot 339
 Swellings caused by jaw tumors 303
 non-osseous jaw tumors 307
 benign non-osseous tumors 307
 histiocytosis X 308
 malignant non-osseous tumors 307
 malignant tumors of
 maxilla 309
 metastatic deposits 308
 multiple myeloma 308
 osseous jaw tumors 303
 benign osseous tumors 303
 malignant osseous tumors 306
 Swellings of jaw 299
 Syphilis 26, 27, 142
 congenital syphilis 28
 early congenital syphilis 28
 late congenital syphilis 28
 latent syphilis 27
 primary syphilis 27
 diagnosis 27
 secondary syphilis 27
 tertiary syphilis 27
 treatment 28
 Syphilitic glossitis 149
 Syphilitic ulcer (gummatous ulcer) 44, 144
 Systemic inflammatory response 9
 endocrine response 9
 immune response 9
 metabolic response 9
-
- T**
- T1 and t2 weighted images 322
 Targeted therapy 234
 Teletherapy 233
 Teratomatous dermoid 112
 Terminologies 317
 Tertiary hyperparathyroidism 292
 Tertiary survey 88
 Tertiary syphilis 27
 Tetanus 30
 clinical features 30
 prophylaxis 31
 treatment 31
 Theater discipline 54
 Thoracic 29
 Thrombocytopenia 65
 Thromboendarterectomy 206
 Thrombophlebitis 225
 Thyroglossal cyst 286
 Thyroid and parathyroid glands 321
 Thyroid antibodies 269
 Thyroid carcinoma 278
 Thyroid eye disease 287
 Joffroy's sign 288
 Moebius sign 288
 Stelwag's sign 287
 von Graefes' sign 288
 Thyroid function tests 268
 isotope scan 269
 thyroid antibodies 269
 Thyroid gland 267
 hypothalamic—pituitary thyroid axis 268
 physiology 268
 surgical anatomy 267
 Thyroid gland 288
 general physical examination 289
 history 288
 local examination of neck 289
 Thyroid neoplasms 277
 benign tumors 277
 clinical features 277
 etiology 277
 malignant tumors 277
 pathology 277

- Thyroidectomy—operative steps 284
 complications of thyroidectomy 285
 hypocalcemia 285
 hypothyroidism 285
 nerve damage 285
 postoperative bleeding 285
 scarring and keloid formation 286
 thyrotoxic crisis 286
 tracheomalacia 286
 wound infection 286
- Thyroiditis 283
 Hashimoto's thyroiditis (chronic autoimmune or lymphocytic thyroiditis) 284
 Reidel's thyroiditis 284
- Thyrotoxic crisis 286
- Thyrotoxicosis 274
 antithyroid drugs 276
 clinical features of thyrotoxicosis 274
 diagnosis for thyrotoxicosis 275
 diffuse toxic goiter 274
 radioiodine 277
 rare causes 274
 signs 275
 specific to Graves' disease 275
 surgery 277
 symptoms 274
 toxic adenoma 274
 toxic multinodular goiter 274
 treatment 276
- Thyrotoxicosis 275
- Tidy wounds 46
- Tissue holding forceps 345
 Allis tissue forceps 345
 Babcock's tissue forceps 345
 Lane's tissue forceps 346
 non-toothed or plain forceps 345
 tongue forceps 346
- Tongue 142
 black or hairy tongue 143
 developmental diseases 142
 congenital fissuring of the tongue 142
 lingual thyroid 142
 tongue tie 142
 geographic tongue (glossitis migrans) 143
 inflammatory lesions of tongue 143
 laceration of tongue 143
 lichen planus 143
 macroglossia 143
 median rhomboid glossitis 142
 ulcers of the tongue 143
 aphthous ulcer 143
 chronic nonspecific ulcer 144
 dental ulcer 143
 malignant ulcer 144
 postpertussis ulcer 144
 syphilitic ulcer 144
 tubercular ulcer 144
- Tongue forceps 346
- Tongue tie 142
- Tonsillectomy 146
- Tonsils 145
 acute tonsillitis 145
 causes 145
 complications 145
 treatment 145
 chronic tonsillitis 145
 treatment 146
 infectious mononucleosis (glandular fever) 147
 clinical features 147
 treatment 148
 parapharyngeal abscess 146
 peritonsillar abscess (quinsy) 146
 clinical features 146
 treatment 146
 retropharyngeal abscess 147
 acute retropharyngeal abscess 147
 chronic retropharyngeal abscess 147
 tonsillectomy 146
 complications 146
 indications 146
 steps 146
- Toothed forceps 345
- Toxemia 21
- Toxic adenoma 274
- Toxic multinodular goiter 274
- Toxicity 236
- Toxoplasmosis 131
- Tracheal dilator 356
- Tracheal intubation 238
- Tracheomalacia 286
- Tracheostomy 178, 180, 182
 aftercare of tracheostomy 182
 aims of tracheostomy 179
 complications of tracheostomy 182
 hemorrhage 182
 intraoperative complications 182
 indications 178
 operation 180
 elective tracheostomy 182
 emergency tracheostomy 180
 surgical anatomy 180
 types of tracheostomy 180
 Tracheostomy instruments 355
 cricoid hook 355
 tracheal dilator 356
 tracheostomy tubes 356
 metallic tube 356
 plastic tubes 356
- Tracheostomy tubes 356
- Transfusion of blood 63
- Transillumination test 108
- Trauma patients 83
 examination of chest and other parts 88
 head and scalp/maxillofacial examination 88
 neck and cervical spine examination 88
 primary survey 83
 airway 83
 breathing and ventilatory support 86
 circulation and hemorrhage control 87
 disability 87
 exposure 87
 secondary survey 87
 tertiary survey 88
- Traumatic gangrene 213
- compartment syndrome 214
 treatment 214
 direct causes 213
 crush injury 213
 pressure sores (bed sores /trophic ulcers) 213
 indirect causes 214
- Traumatic injuries 320
- Traumatic ulcer 42
- Treatment of neck nodes 157
- Treatment of primary tumor 153
- Trench foot 215
- Trendelenburg procedure 224
- Triage 82
- Trigeminal nerve 193
- Trigeminal neuralgia 194
 investigations 194
 treatment 194
- Trocar and cannula 353
- Tropical ulcer (phagedenic ulcer) 44
- True neuromas 91
- Tube drain 357
- Tubercular ulcer 44, 144
- Tuberculosis 22
 clinical features 22
 intestinal tuberculosis 23
 lupus vulgaris 23
 milary tuberculosis 23

pulmonary tuberculosis 22
 tuberculosis of bone and joint 23
 tuberculous lymphadenitis 22
 investigations 24
 modes of spread 22
 by ingestion 22
 droplet infection 22
 multiple drug resistance (MDR)
 tuberculosis 25
 causes 25
 directly observed treatment (DOT)
 for tuberculosis 26
 surgical treatment 26
 treatment 25
 other measures 25
 side effects 25
 treatment of cold abscess 26
 Tuberculous lymphadenitis 22,368
 Tubular bones 244
 Tubulo-embryonic dermoid 113
 Tumor necrosis factor-alpha 10
 Tumors 89
 Tumors of larynx 177
 benign tumors 177
 malignant tumors 177
 advanced laryngeal tumors 178
 classification 177
 etiology 177
 incidence 177
 investigations 178
 treatment 178
 vocal rehabilitation after
 laryngectomy 178
 Turban tumor (cylindroma) 95

U

Ulcer 36, 38, 39
 classification 38
 clinical examination of an ulcer 39
 history 39
 general examination 40
 investigations 40
 life history of an ulcer 39
 local examination 39
 regional examination 40
 systemic examination 40
 treatment 41
 Ulcerative stomatitis (Vincent's angina)
 141
 Ulcers of tongue 143
 Ultrasonography 315

Doppler ultrasound 317
 advantages of ultrasound 317
 drawback of ultrasound 318
 terminologies 317
 principle 315
 Ultrasound for head and neck lesions
 318
 Uncomplicated closed fractures 247
 Underlying cause 42
 Unfit patient 7
 Untidy wounds 46
 Urethral catheterization 361
 Urethral catheters 359

V

Vagus nerve 194
 Varicose ulcer 225
 Varicose veins 221
 calcification 226
 complications of varicose veins 224
 dermatitis 225
 foot deformity 226
 hemorrhage 226
 lipodermatosclerosis 225
 Marjolin's ulcer 226
 new surgical techniques 224
 operative techniques 224
 stripping of long saphenous
 vein 224
 stripping of short saphenous
 vein 224
 Trendelenburg procedure 224
 periostitis 226
 pigmentation 225
 primary varicose veins 221
 secondary varicose veins 221
 clinical examination 221
 clinical features 221
 investigations 222
 treatment 223
 thrombophlebitis 225
 ulceration 225
 surgical treatment 225
 treatment of varicose ulcer 225
 Varicose veins 224
 Vault and skull base lesions 320
 Venesection cannula 358
 Venous cut down 359
 Venous gangrene 213
 Venous system 220
 Venous ulcer 42

Viral infections 34
 Vitamin K deficiency 65
 Vocal cord palsy 176
 clinical features 176
 etiology 176
 investigations 177
 treatment 177
 Volkmann's scoop (curette) 355
 von Graefes' sign 288
 von Willebrand's disease 66

W

Warthin's tumor 168
 Water and electrolyte balance and
 imbalance 71
 disturbances in electrolyte balance 72
 hyperkalemia 72
 hybernatremia 72
 hypokalemia 73
 hyponatremia 72
 disturbances in water balance 71
 postoperative fluid therapy 74
 period of therapy 74
 types of IV fluids 74
 Water balance 71
 Well's arterial clamp 348
 Wound dressings and bandages 363
 Wound healing 52
 Wound infection 15
 Wounds 46, 50
 complications of wound healing 52
 definition 46
 tidy wounds 46
 untidy wounds 46
 examination of wounds 50
 management of facial wounds 51
 medicolegal aspects of wounds 51
 dangerous to life 51
 grievous injury 51
 simple injury 51
 treatment of wounds 50
 types of wound 46
 avulsion wounds 48
 crushed wounds 49
 incised wounds 48
 lacerated wounds 48
 punctured wounds 48
 wound healing 49
 phases of wound healing 49
 repair of surface epithelium 49